Proceedings
of the
Third
Symposium
on the

Prevention of Handicapping Conditions of Prenatal and Perinatal Origin

1981

Sponsored By:

Alberta Social Services and
Community Health
The Canadian Institute of Child Health
Glenrose Provincial Hospital

Alberta
SOCIAL SERVICES
AND COMMUNITY HEALTH

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Prevention of Handicapping Conditions of Prenatal and Perinatal Origin

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FOREWORD

The Third Symposium on the Prevention of Handicapping Conditions, which was held at the Glenrose Hospital, Edmonton, Alberta, followed two very successful programs. This year The Canadian Institute of Child Health and the Glenrose Hospital, as part of their promotion of the International Year of Disabled Persons, were co-sponsors with Alberta Social Services and Community Health.

The purpose of this symposium was to bring together professionals interested in new developments in the prevention of handicaps. We were indeed very fortunate to assemble a distinguished panel of twenty speakers which included six international authorities and sixteen local experts.

There were 270 people formally registered, with some participants attending either October 28 or October 29. Those who attended were from a variety of disciplines including physicians, nurses, physiotherapists, occupational therapists, psychologists, teachers, social workers and special educators.

In order to continue the goal of public involvement, to raise awareness about new programs and the role of the public in prevention, the symposium opened with a public address on the topic "Newborn Care and the Prevention of Cerebral Palsy", attended by over 250 people. The address was sponsored by The Canadian Institute of Child Health as the John T. Law Lecture. John Law made many contributions in the health field, and one of the priority areas he identified was the prevention of handicaps. For this reason, the Symposium for the Prevention of Handicapping Conditions of Prenatal and Perinatal Origin was an appropriate setting for a John T. Law Memorial Lecture. Professor Sir Peter Tizard, a prominent paediatrician from Oxford, England, was featured as keynote speaker.

Other objectives of the Third Symposium were to encourage the sharing of ideas of different health disciplines and, by means of discussion and presentation of information, to identify obstacles to primary prevention of handicaps. As a result, a list of recommendations was made by the moderators and speakers, and another by the participants. These recommendations appear in Appendices A and B.

Other goals were to encourage the involvement of treatment personnel in prevention and to promote an atmosphere for change by highlighting the potential for improvement in measures to prevent handicaps.

The Honourable Bob Bogle, Minister of Alberta Social Services and Community Health, opened the symposium with an inspirational address, and we would like to thank him for his continued support. We would like to express our gratitude to the members of the Program Planning Committee and the Steering Committee, as it was their time and effort that made the symposium possible. The valuable contribution of the moderators is also greatly appreciated.

The publication of this monograph was made possible through the co-operation of the speakers in providing us with copies of their manuscripts. Owing to this co-operation, there is a wealth of information contained in these proceedings. Following the precedent set in last year's monograph, little or no editing was done to the manuscripts.

We would also like to thank Carol Althouse, Helen Dyksley and their staff at Word Processing for their patience and help in preparing these presentations, Peter Tong in Public Communications for his time and assistance in producing this monograph, the reviewers for their advice, Min Feldman, Jo Anne Dodds and Janis Hudson for their invaluable assistance in collecting and compiling all the articles.

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August 1982

TABLE OF CONTENTS

Page

Foreword iii

Highlights of Opening Address 1

Presentations:

JOHN T. LAW LECTURE
NEWBORN CARE AND THE PREVENTION OF CEREBRAL PALSY
Professor Sir Peter Tizard 3

FACTORS INFLUENCING BRAIN DEVELOPMENT
Dr. Jonathan Wigglesworth 17

MATERNAL SMOKING DURING PREGNANCY AND CHILD DEVELOPMENT:
A REVIEW, AND THE METHODOLOGY FOR A NEW STUDY OF OVER
10,000 REPRESENTATIVE FIVE YEAR OLDS IN GREAT BRITAIN
Dr. David Rush 31

INTRAVENTRICULAR HAEMORRHAGE AND HYPOXIC BRAIN INJURY
Dr. Jonathan Wigglesworth 55

FACTORS AFFECTING OUTCOME IN HYPOXIC-ISCHEMIC ENCEPHALOPATHY
IN TERM INFANTS
Dr. Neil Finer 65

DEVELOPMENT OF HIGH RISK CHILDREN IN THE FIRST YEAR OF LIFE
Dr. Reginald Sauve 81

MYOMETRIAL EFFECTS ON FETAL DEVELOPMENT
Dr. Peter Nathanielsz 95

ASSESSMENT OF THE FETUS AT RISK
Dr. Bob Richards 107

ASSESSMENT FOR SENSORI-MOTOR STIMULATION
Dr. Mervyn Fox 125

THE ANATOMIC AND PHYSIOLOGIC BASIS OF NEUROLOGIC DEVELOPMENT
IN THE PERINATAL PERIOD
Dr. Harvey Sarnat 157

RETROLENTAL FIBROPLASIA - A HANDICAPPING CONDITION OF PERINATAL
ORIGIN
Dr. Warren Hindle 177

Page

THE DENVER APPROACH TO DEVELOPMENTAL SCREENING AND DIAGNOSIS OF INFANTS AND PRE-SCHOOL CHILDREN
Dr. William Frankenburg 181

PART 1: NEW ASPECTS IN THE RECOGNITION AND MANAGEMENT OF CEREBRAL PALSY
Dr. Charlene Robertson 199

PART 2: THE EARLY MANAGEMENT OF GROSS MOTOR DIFFICULTIES IN CEREBRAL PALSIED CHILDREN
Dr. Joe Watt 211

EARLY IDENTIFICATION OF CHILDREN WITH COMMUNICATION DISORDERS
Ms. Jo Anne Mahood 219

THE "HIGH RISK" CONCEPT REVISITED
Dr. William Frankenburg 231

FAMILY RELATIONSHIPS AND INFANT MENTAL HEALTH DISORDERS: SOME ISSUES AND OPPORTUNITIES FOR PREVENTION
Dr. Sally Provence 241

MATERNITY/NEWBORN CARE - PSYCHO-SOCIAL CARE IS GOOD PREVENTION
Mrs. Shirley Post 259

HEALTHY BABIES - EVERYONE'S BUSINESS: THE MANITOBA COMMUNITY TASK FORCE ON MATERNAL AND CHILD HEALTH
Mrs. Agnes Hall 275

THE LAW AND REPRODUCTIVE CARE
Mrs. April Katz 283

ASSESSMENT OF THE NEUROLOGICALLY-IMPAIRED FULL TERM INFANT
Dr. Husam Darwish 289

TO WHAT EXTENT CAN WE PREVENT MENTAL RETARDATION?
Professor Sir Peter Tizard
Professor Tizard sends his regrets that due to a serious illness he was unable to send his second paper in time for publication.

Page

Appendices

A. Recommendations of Speakers and Moderators
Dr. Graham Chance
University of Western Ontario 305
B. Recommendations of Participants 307
C. Program Planning Committee 309
D. Steering Committee 311
E. Moderators 313
F. Speakers 315
G. Biographies 317
H. Participants 323
I. Program of the Third Symposium 331

HIGHLIGHTS OF OPENING ADDRESS

Delivered by
Honourable Bob Bogle
Minister of Social Services and Community Health
Edmonton, Alberta

The opening address was delivered by the Honourable Bob Bogle, Minister of Alberta Social Services and Community Health. The Minister expressed enthusiastic support for prevention of handicaps, and was indeed pleased to see that within the short span of less than three years the symposium had gained sufficient stature to warrant being held in a hospital facility that was specially designed for the treatment of handicapped children of all ages.

He went on to describe some preventive projects that are currently underway in the province of Alberta such as: "The Rubella Prevention Program", a public awareness program to ensure that susceptible women will be made aware of the risk of this infection during pregnancy and the measures that they may take to ensure protection; and the "Hereditary Diseases Program", an outreach service to provide families throughout the province with genetic counselling. This service commenced in 1980 and is now available through the health units.

The Minister was pleased and satisfied with the progress to date, and it was felt that we could count on his continued support for the implementation of programs for handicapping conditions that to a large extent can be prevented.

SPEAKERS

Standing left to right: Dr. Reginald Sauve, Dr. Warren Hindle, Dr. Robert Haslam, Dr. J.K. Martin, Dr. Harvey Sarnat, Dr. Peter Nathanielsz, Dr. Jonathan Wigglesworth, Dr. Wm. Frankenburg.

Sitting left to right: Dr. Mervyn Fox, Professor Sir Peter Tizard, Dr. Sally Provence, Mrs. Agnes Hall, Mrs. April Katz, Mrs. Shirley Post, Dr. David Rush.

Missing from picture: Honourable Bob Bogle, Dr. Neil Finer, Dr. Bob Richards, Dr. Husam Darwish, Dr. Charlene Robertson, Ms. Jo Anne Mahood, Dr. Graham Chance, Mr. Lloyd Wilson, Dr. Joe Watt.

THE JOHN T. LAW LECTURE

"NEWBORN CARE AND THE PREVENTION OF CEREBRAL PALSY"

Professor Sir Peter Tizard
Department of Paediatrics
John Radcliffe Hospital
Headington, Oxford

P. Tizard

First may I say how very grateful I am to have been asked to give the second John T. Law Memorial Lecture. The original invitation came from my old friend and ex-colleague, Professor David Schiff, but a number of other individuals and organizations have been involved and to all of them I am most grateful. The organizations include Alberta Social Services and Community Health, the Canadian Institute of Child Health and the Glenrose Provincial Hospital. The individuals include Dr. Paton, Mr. Wirsig, Dr. Beaudry and Dr. Etches, another ex-colleague.

The doctors in the audience who know the subject well, and especially those who know me well, will already have realized that I am going to say nothing startling or new or, indeed, very encouraging about the prevention of cerebral palsy in the neonatal period. One can say at the outset that cerebral palsy will never be wholly prevented since some cases, albeit a minority, are the result of accidents and even occasionally of mismanagement round about the time of birth, and, while one can reduce the risk of accident and mismanagement, it is impossible to eliminate completely the effects of chance or of human error. What I propose to do in the time allotted to me is as follows. Firstly, I intend reporting the work of others who have shown conclusively that the occurrence of cerebral palsy has declined in the last two or three decades. Secondly, I will refer to modest hopes of further improvement in the future as the result of a more general application of already accepted ideas. Thirdly, I will describe briefly some current research which may possibly hold out greater hopes in the long term.

Meaning of Term 'Cerebral Palsy'

What do we mean when we use the term cerebral palsy? The popular expression in England is 'the spastic child', but not all children with cerebral palsy are spastic in the medical sense of the term. Cerebral palsy is a convenient term under which to categorize all defects of movement or posture caused by damage to the brain originating before birth, at birth or in the early post-natal years. By convention, it does not include defects of movement resulting from progressive disorders of the brain, such as tumours or rare degenerative diseases. But the definition I have given you would not only include the most severely affected children, those who are completely dependent on others for the most trivial aspects of everyday life - and this is all the more tragic when accompanied, as it often is, by normal intelligence - but, if you pause to reflect on my definition, it will also include at the other extreme the clumsy child! And clumsiness, although undoubtedly a minor handicap, cannot be regarded as one of life's greater tragedies. Thus one of the difficulties in counting up cases of cerebral palsy in a community lies in the fact that no one has really agreed exactly where it begins or ends. But it is fair to say that most paediatricians and others involved in the care of cerebral palsied children have a reasonable working practice for the diagnosis of cerebral palsy.

Prevention of Cerebral Palsy

At this point in my talk I want to point out something that is obvious to all doctors, but not obvious to other intelligent individuals - those whom we doctors arrogantly refer to as the laity! It is sometimes possible to say with certainty that had one not taken such and such an action a newborn baby would have died. But it is very seldom possible to say with any degree of certainty that without a particular treatment - preventive or curative - a baby would have survived with cerebral palsy. One can practically never say 'but for my intervention this baby would have had cerebral palsy; now he is going to be normal!'

As a rule it is impossible to diagnose cerebral palsy in a newborn baby. This is because the brain, or that part of it involved in conscious thought and voluntary movement, plays but a small part in movements of the limbs at birth and, thus, while one may suspect that cerebral palsy will develop, one has to await the appearance of signs of cerebral palsy after weeks or even months, as the brain gradually assumes its powers of maintenance of posture and of voluntary movement.

Epidemiology

Since one can very seldom be certain that one has saved a newborn baby from cerebral palsy, a study of the prevention of these conditions must be an epidemiological exercise, i.e., done on a population basis. This means having a clear definition of exactly what constitutes cerebral palsy and the ability to trace all cases in a given community. This latter is a very exacting and difficult task, especially in large countries and in an age when families tend to move from place to place. To make any sense of the numbers game and reliably to discover trends, one must be dealing with relatively large numbers. Nevertheless, this has been achieved with reasonable accuracy in certain countries or parts of countries with relatively stable populations and well-defined borders.

Changes in cerebral palsy over a 25-year period are shown in three such studies: the first from Dr. Paul Glenting in the whole of Denmark east of the Little Belt, the second from Professor Bengt Hagberg in the Gothenburg district of Sweden and the third from Dr. Fiona Stanley in Western Australia. Each of these studies demonstrates a pretty steady fall in the prevalence of cerebral palsy as a whole and in each of its main types over the past two decades.

To what can we attribute this significant and welcome improvement? Modern obstetric and neonatal paediatric practices and techniques have led to an ever-increasing survival of babies who, in the recent past, would have died. Our figures from Oxford provide an example. The decline in perinatal mortality rates - that is, the occurrence of stillbirth in late pregnancy or death in the first postnatal week - has led to two apparently contradictory arguments. The first is that modern methods of care of the prematurely born or ill or asphyxiated full-term baby are leading to the survival of brain damaged children who in the past would have died. The second is that the very methods which have led to the survival of previously doomed babies are also leading to the survival without brain damage of babies who in the past would have survived with brain damage.

The fact is that there is no clear-cut relationship between perinatal mortality and the incidence of survival with handicap. Both the statements I adduced as contradictory are true. It is the balance between the two that we need to know, and as a generalization one can say with assurance that improvements in perinatal mortality have not been accompanied by an increase in the incidence of cerebral palsy. Again, I quote Professor Bengt Hagberg. He is just about to publish a paper in which he compares perinatal mortality and the incidence of cerebral palsy in the years 1966 to 1970 and 1971 to 1975. There has been a marked decrease in perinatal mortality in Sweden, accompanied by a fall followed by a very slight increase in recent years in the incidence of cerebral palsy. From his figures, Professor Hagberg concludes that 1 out of 40 saved lives acquired a cerebral palsy syndrome.

To what is this undoubted improvement in both perinatal mortality figures and - over the past 20 or 30 years - in the occurrence of cerebral palsy to be attributed? Many factors must be involved, especially, perhaps, rising living standards in the Western world since perinatal mortality has always shown a socio-economic class bias. Then one can point to very definite improvements in the medical management of pregnancy, especially the concentration of care into larger maternity hospitals with the elimination of small maternity units and, rather sadly in my opinion, the almost complete disappearance of home delivery in England. The concentration of care has been accompanied by increasingly effective methods of detecting in early or mid-pregnancy those mothers who are at risk of early or abnormal deliveries, often permitting the application of preventive measures. In respect of neonatal paediatrics, improvements have in part been due to the application of the complex methods of intensive care, but probably they are even more attributable to relatively simple measures more universally applied. I refer to the effective resuscitation at birth of the baby who does not immediately breathe, to the importance now - but only in recent years - attached to maintaining a baby's body temperature, keeping it warm, the importance as well as the hazards of early and adequate feeding and the realization that handling an ill newborn baby has to be kept to an absolute minimum since even what appear to us as quite innocuous changes in the environment - such as the ward telephone ringing - may cause deterioration in an ill baby.

I have referred to the importance of concentrating the antenatal care of mothers whose unborn babies are at risk of early or complicated delivery. Concentration of care is equally important in effective intensive care of the prematurely born or ill term baby. It is true that minimal standards for any maternity unit should include facilities for ventilating the lungs of a baby who is not breathing or is breathing inadequately, in the hope of a quick recovery or of early transfer to a neonatal intensive care unit. But there is a temptation for doctors and nurses in small maternity units to keep such babies under their care - a temptation that is easily understood for a number of reasons, including the wish not to separate mother and baby in different hospitals. However, artificial ventilation of the newborn baby is a complex procedure, and one in which success is determined to a large extent by the experience of the doctors and nurses concerned. This is relevant to cerebral palsy as well as to survival as I shall shortly explain.

With growing impoverishment in the United Kingdom, we are once more becoming dependant on private charity for the provision of essential equipment once readily obtainable through the National Health Service. A forceful and determined young paediatrician can play upon local pride and generosity in equipping his newborn nursery beyond the point justified by his training and continuing experience and by the staffing of his unit. It should be borne in mind that giving complex equipment to small units may at times be worse than useless.

When one begins to look more closely at the means of prevention of cerebral palsy, it is important to realise that it is not one disorder.

Classification of the cerebral palsies has been the subject of windy controversy for years, and well it might, since pathological processes do not respect anatomical boundaries in the brain, let alone functional boundaries to the extent that these exist. Defects of the brain, other than those causing disorders of movement, may co-exist; mental retardation, defects of sensation - sight, hearing and discriminatory sensation in the fingers - and, sometimes, epilepsy. The defects of movement alone are variable. They can be broadly classified into spasticity (muscular stiffness), dyskinesia (involuntary movements) and ataxia (unsteadiness). More than one of these defects of movement may co-exist in the same child. The Table shows my simplified classification of the cerebral palsies and its only claim to originality lies in the fact that it is not precisely like anyone else's. The dyskinetic cerebral palsies involve all four limbs and trunk, head and organs of speech, and are classified according to the type of abnormal movement; the spastic cerebral palsies according to distribution to the limb or limbs that are involved. The distinguished British paediatrician, Dr. Philip Evans, gave a learned, useful and amusing talk on the classification of the cerebral palsies to a meeting at the Royal Society of Medicine in 1948. He said, and I quote, "starting with a monoplegia, a one stroke, we go on to a half stroke, which is twice as extensive as a monoplegia; a diplegia or two stroke is actually four times as extensive as a one stroke but affects twice as many limbs as a half stroke."

Incidentally, the first description of some forms of cerebral palsy was made by a British obstetrician called William Little in 1843. He was the first person to attribute these disorders to abnormal delivery. The connection may seem obvious now, but Little's discovery was a shrewd clinical observation because, as I have said, defect of movement is not apparent at first in infants who, months later, show unmistakable signs of cerebral palsy. Thus the connection between events at birth and what subsequently appeared was far from obvious. Fifty years after William Little's publication saw the next really outstanding contribution to the study of cerebral palsy by, and this may surprise you, Sigmund Freud. He was a neurologist and neuropathologist before turning to psychiatry. His only disservice to the subject was his coining of the term diplegia which has led to much misunderstanding. In cases of diplegia the legs are worse affected than the arms - an extension of cerebral paraplegia; in hemiplegia or double hemiplegia the arms are worse affected than the legs and there is a convincing neuropathological basis for both clinical findings.

You are probably all familiar with what is meant by the term athetosis. Choreoathetosis is a form of dyskinetic cerebral palsy that has almost completely disappeared in recent years, because it was associated with severe jaundice in the newborn, usually secondary to rhesus haemolytic disease, and we can now prevent the latter and effectively treat the former by exchange blood transfusion. As a great rarity, congenital chorea exists. This condition, not severely disabling and associated with normal intelligence, is probably inherited from parent to child.

The term dystonia has been used by Dr. Kyllerman from Goteborg for what is usually the most serious form of cerebral palsy, in which there are not so much involuntary movements as involuntary assumption of weird postures, preservation into childhood of reflex activity normally seen in the newborn and very severe disorders of movement. When this condition is accompanied, as it sometimes is, by normal intelligence, it is perhaps the most tragic form of all the cerebral palsies.

Now I have already told you that choreoathetosis was caused by severe jaundice in the first few days after the birth. There is no other form of cerebral palsy in which the cause and effect relationship is so clearly established. It is usually the case that we cannot give parents any clear explanation of why their child has developed cerebral palsy. Take hemiplegia for instance; this is sometimes caused by a stroke occurring in a previously perfectly healthy child in whom, usually in the course of tonsillitis, a clot has formed in the carotid artery and has cut off the blood supply to the major part of one half of the brain. However, most cases of hemiplegia do not occur this way. Some cases are complications of birth asphyxia or injury but many more appear to originate before birth and we do not know the nature of the pathological processes involved.

However, there are certain associations that are commonly encountered, and I propose to tell you about two of them in order to introduce some recent research being carried out in my department. The first is that of intraventricular haemorrhage which, when causing cerebral palsy, usually produces paraplegia or diplegia. The second is severe intrapartum asphyxia leading typically to dystonia, although other forms of cerebral palsy may result.

Now I do not propose to enter into lengthy descriptions of either, especially not the first because this is the particular province of Dr. Jonathan Wigglesworth. Suffice it to say that bleeding into the ventricles of the brain does not appear to be due to injury, that it can now be diagnosed reliably in life by the use of CAT scans or ultrasound and that while many babies who have had small haemorrhages survive to be entirely normal a minority are slow to sit up, to stand and to walk and are found after a few months to have spastic legs. The arms and hands are, as a rule, but little affected. These children are not usually mentally retarded, have no disorders of sensation and are not likely to suffer from fits in later childhood. But a child so affected will never be good at games and will never dance gracefully.

(A retrospective analysis of William Little's 48 cases reveals an association to which he did not draw attention - namely, that when the children had 'rigidity' confined to the legs they had nearly all been born prematurely but otherwise normally, whereas when the arms were also involved these children had nearly all been born at term but had suffered birth asphyxia.) Now Dr. Wigglesworth's pathological studies have suggested to him that alteration in the flow of blood through the carotid and vertebral arteries into the brain - cerebral blood flow - is likely to be the cause of these haemorrhages. A fall in cerebral blood flow may damage a certain area of the brain; a sudden increase in flow will cause bleeding into this area and subsequent rupture of the clot through into the ventricles. In order to understand the circumstances that lead to these dangerous alterations in blood flow to the brain, we must of course be able to measure blood flow, and this is very difficult to do. Various methods have been employed that involve needling the baby and/or injecting radio-active substances. But this is not the sort of procedure to which one would subject one's own newborn baby - surely the best guide for ethical practice - and one must know what happens in the normal baby to be able to assess the significance of findings in an ill baby. One entirely harmless method was devised a few years ago by Professor Kenneth Cross of the London Hospital Medical School and has been applied by Dr. Finer and Dr. Etches in Edmonton. It depends on the fact that the skull bones of a newborn baby are very flexible, and that one can sometimes see the newborn baby's head pulsating in time with heart beats. If one were to put a tape measure round the baby's head and occlude the jugular veins for two or three seconds, the head would enlarge very slightly and a measure of its enlargement would give a guide to the amount of blood entering the head and not immediately leaving it. In practice, this is done with a mercury strain gauge. When the tube is elongated, the electrical resistance in it rises. This change in resistance can be measured, and the resultant increase in the volume of the head calculated.

However, there are three disadvantages attached to this method. The first is that it is not particularly accurate for a variety of reasons that I will not detail. The second is that it means disturbing the baby - not unfair in the case of a perfectly healthy baby but likely to be harmful when a baby is very ill. Thirdly, one can only obtain intermittent measurements and, unless one can obtain continuous measurements of blood flow to the brain, one is likely to miss the very circumstances in which blood flow is suddenly altered - increased or decreased. One method that we have been examining in our department is that of electrical impedance plethysmography under the guidance of Dr. Peter Rolfe, Head of our Bio-engineering Unit. Impedance means the resistance to flow of an alternating electrical current, and this resistance varies with changes in conductive elements, such as the cerebro-spinal fluid and the blood in the brain. A small high-frequency current (2mA, 100 kHz) is passed through the head between one electrode placed on the centre of the forehead and another electrode at the back of the head. This is an entirely harmless and painless procedure, and we have, for instance, satisfied ourselves that it neither produces electrical interference on the electroencephalogram of the baby nor alters the appearance of the electroencephalogram - i.e., brain waves are unaltered. The other two electrodes are used to measure the voltage across the head and, hence, by Ohm's law transcephalic impedance is derived.

We record two impedance measurements. The basal impedance is a large, slowly varying value of 23-35 ohms which reflects major changes in the resistivity of structures within the head, such as fluids in cases of hydrocephalus, large intraventricular bleeds and cerebral edema - water-logging of the brain. A second much smaller impedance measurement varies in time with the pulse, and to a lesser extent with respiration, and is usually between 20 and

40 milli-ohms. We have reason to believe that the size of this pulse is proportional to the amount of blood entering the head with each heart beat. The electrodes may be left in place for many hours without disturbing the baby in any way. We have attempted to validate this method by comparing changes in electrical impedance with changes in strain guage resistance - using the jugular compression method. Dr. Kate Costeloe and Dr. Diane Smythe administered 2% CO_2 or 100% O_2 for short periods to 10 healthy term babies - with parental permission and in the presence of their mothers. As one would expect, inhalation of CO_2 usually showed an increase in apparent cerebral blood flow by both methods; inhalation of O_2 a decrease, but a linear relationship could not be demonstrated.

However, we have good reason to suppose that cerebral electrical impedance does give an indication of blood flow into the baby's head, although it has not been possible to calibrate, to quantify, these measurements. Nevertheless, we are confident that changes in electrical impedance will reflect changes in cerebral blood flow.

Here are two examples of measurements made in ill babies. One of the complications of ventilator treatment of prematurely born babies is that air may leak out of the lungs into the space between the lungs and the chest wall with serious consequences to respiration unless the air is promptly removed by needling the chest. This is the condition known as a pneumothorax, and Professor Osmund Reynolds and his colleagues at University College Hospital in London have already shown that the pneumothorax is associated with the development of intraventricular haemorrhage.

Cerebral electrical impedance was recorded in a prematurely born baby with respiratory distress who developed a pneumothorax. As the baby's condition deteriorated, apparent cerebral blood flow fell and, after the chest was drained of air, it returned to its previous value.

Dr. Wigglesworth has suggested that administration of alkalis to a baby who is acidotic with respiratory failure may precipitate an intraventricular haemorrhage. We have demonstrated a surge in apparent cerebral blood flow when an alkali (THAM) was injected.

I now turn to the other topic, that of severe birth asphyxia and the various forms of cerebral palsy that can ensue. The first point I wish to make is this. Three associations are known. Intrauterine growth failure - smallness for dates, as it is generally known - increases the risk of birth asphyxia. Severe birth asphyxia may lead to cerebral palsy. But Professor Bengt Hagberg has shown that cerebral palsy as a whole is associated with smallness for dates. I have enquired from Professor Hagberg what proportion of small-for-dates babies who subsequently developed cerebral palsy had also suffered birth asphyxia, and he cannot give me this information, but it is worthwhile remembering that intrauterine growth failure may be associated with brain damage before the child is born rather than being the consequence of asphyxia at birth.

My next point is that it is usually worthwhile trying to resuscitate an asphyxiated newborn baby even when the baby appears to be dead, i.e., has no discernable heart action, particularly when a baby known to have been alive shortly before delivery is quite unexpectedly born dead. At one time, we thought that this would inevitably lead to serious and permanent brain damage, but this is by no means necessarily the case. The fetus and newborn baby is much less likely to suffer permanent brain damage through suffocation than is an older child or adult. The first person to show that this was true in the experimental animal was Dr. Robert Boyle in 1670. There have been several follow-up studies of babies who either took more than 30 minutes to start regular breathing after birth or who had no spontaneous heart action at birth. Of those who survived the neonatal period - and about half of them died - the majority turned out to be normal. But some such babies, however rapidly and skillfully resuscitated, especially those who are known to have been in trouble during labour, do develop a very serious illness soon after birth caused by damage to brain cells and edema (waterlogging) of the brain - an illness which is accompanied by disturbances of consciousness and by fits. Once this has happened the outlook in terms of brain development is very poor. We have recently been trying the effect of barbiturate treatment - using a barbiturate called thiopentone - in an attempt to prevent or ameliorate brain damage in babies who have been severely asphyxiated at birth.

One of the consequences of damage to brain cells is that the brain becomes waterlogged and this, so-called edema, can in itself cause further damage. Our treatment with thiopentone has proved disappointing. We have shown quite conclusively that we can prevent an increase in edema and, indeed, shown that edema recurs when thiopentone treatment is stopped, even as long as 10 days after birth. It is true that in experimental animals administration of barbiturate drugs before suffocation will protect the animal's brain. We have recently had one highly successful case of a boy of eight months of age whose heart stopped for eight minutes during an operation and who, in spite of a brain illness but probably because of the thiopentone treatment, has made a complete recovery. But the problem with the newborn babies we have treated is that each has shown evidence of having been asphyxiated for some time before birth, evidence either from the record of the fetal heart rate or from the finding that the blood was acid at birth, a sure indication of prolonged asphyxia. As I have already told you the baby who is found unexpectedly dead at birth, having been known to have been in good shape before delivery, if resuscitated efficiently has a very good outlook. Perhaps what the thiopentone trial has shown us is that we should not try to resuscitate babies apparently dead at birth when they have been known to have been in difficulties before delivery or that we should not continue to resuscitate a baby who does not breathe within 30 minutes of delivery.

In fact this was the message conveyed by a reanalysis of the findings of one study - that of Dr. Hilary Scott. Cardiac arrest at birth did not necessarily imply serious brain damage. Prolonged delay in establishing normal breathing did.

I have given you two examples of neonatal research, chosen simply because they emanate from my department, one of which might eventually help to prevent cerebral palsy. Of much greater importance is obstetric research into the causes and prevention of premature birth, the causes and prevention of disturbances of growth in the fetus and the causes and prevention of asphyxia in labour. But my task was to talk about the newborn care and the prevention of cerebral palsy. I should perhaps have said earlier that it is rather less than half of all cases of cerebral palsy that seem to have their origin round about the time of birth, about half of all cases having suffered brain damage earlier in pregnancy and about one in ten after birth. Thus, however effective is newborn care, improvements in newborn care alone would not solve the problem.

I now want to digress for a moment and tell you of circumstances in England that are likely to lead to an increase, I hope only a small increase, in the prevalence of cerebral palsy. I have under my care a baby born two months ago, who was known to be in difficulties before delivery and who was born apparently dead. One of our residents resuscitated the baby, but it became immediately evident that there were signs of brain disturbance and we gave the baby the full treatment with the drug called thiopentone. After about five days, it became evident that the treatment had been unsuccessful and when the baby was two weeks' old I added up 10 or 11 factors each of which taken separately indicated an extremely poor outlook for the baby and, taken together, led to the inevitable conclusion that, if he survived, he would survive with the most severe form of cerebral palsy. Shortly afterwards, it became possible to remove the endotracheal catheter, the tube in his windpipe, through which his lungs were being ventilated since he was then breathing spontaneously and effectively. In the past, I would have told my residents that had this baby stopped breathing again he should not have been resuscitated.*

Going abroad cuts one down to size as far as the importance of one's own country is concerned. I have searched in vain for information about England in the newspapers. But, from time to time bad news is reported and there have recently been two cases in England in which the law has become entangled with the practice of neonatal paediatrics. I consulted my medico-legal adviser about the baby I have just described to you and, after some consideration, he told me that, if I pursued the course of action I thought proper, I would be likely to find myself on a murder charge, although I probably would not be convicted! While nearly every doctor regards it as his primary duty to keep his patients alive, however severely disabled, there are occasions in which our practice does not follow that principle. If any member of the audience feels that there are no circumstances in which a doctor should allow his patient to die, then there are no grounds on which he and I can argue the case.

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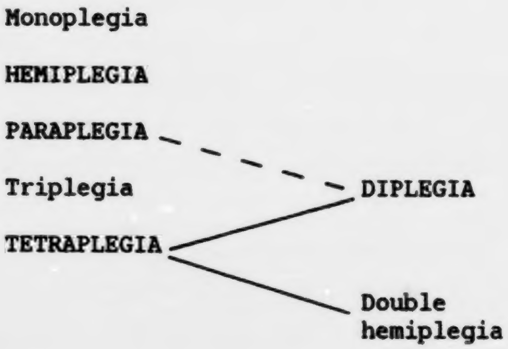
*The baby has survived, is now seven months old and there has been no growth in head circumference - and thus of his brain - since birth.

I have given you but a patchy account of the changes in neonatal care and their influence on the occurrence of cerebral palsy. I want to end on a note of hope. It is true that the more the prevalence of cerebral palsy is reduced, the harder it becomes to reduce it further, but there are still very obvious ways in which we could apply more widely what is already known and still obvious lines of research to be pursued. I feel optimistic, and I hope I will have conveyed my optimism to you.

THE CEREBRAL PALSIES

SPASTIC



- Monoplegia
- HEMIPLEGIA
- PARAPLEGIA
- Triplegia
- TETRAPLEGIA

DIPLEGIA

Double hemiplegia

DYSKINETIC (bilateral)

- ATHETOSIS
- Choreoathetosis
- DYSTONIA

Ataxic

Table 1

FACTORS INFLUENCING BRAIN DEVELOPMENT

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Introduction

In a conference devoted to prevention of handicap any consideration of factors influencing brain development must be directed towards discussion of the balance of influences which may promote or mitigate brain damage in the fetal and perinatal period. Whereas all organ development is complex, that of the brain involves integration of an enormous number of different processes which any brief description can represent only in crude outline. A developmental neurobiologist viewing brain development does so in the light of an extensive knowledge of one area of the process which he may stress at the expense of others. As a perinatal pathologist, I have to see the brain merely as one organ among many which may be malformed or suffer functional impairment in the perinatal period, and can thus give only an impressionistic view of the processes of brain development. This does, however, allow freedom to speculate on the ways in which brain development may be influenced for better or worse, unfettered by the minutiae of an excess of knowledge.

This paper is offered in the hope that it may encourage a flexible and imaginative approach to the problems of brain development among both clinicians and basic scientists.

Scheme of Normal Brain Development

Perinatal brain development should most logically be considered as a single, continuous process involving structured and functional integration at all stages. It is, however, easier to present in terms of sequences of different types of structural or functional development.

A. The sequence of gross structural development

The initial formation of the neural tube takes place during the third or fourth week after conception.(1) The different regions of the brain are indicated from an early stage by a series of flexures. The midbrain (mesencephalic) flexure develops first, followed by flexures delineating the brain stem (cervical and pontine flexures). The cerebral hemispheres become recognizable in the form of paired cerebral vesicles by the end of the fifth week. The cerebellar primordia appear relatively late and only fuse in the midline near the end of the embryonic period of about nine weeks.

Through prenatal life, development of the cerebral hemispheres and cerebellum lags behind that of the basal ganglia, thalamus, midbrain and brainstem. During the fetal period, however, the cerebral hemispheres and cerebellum grow faster than the midbrain and brainstem and thus gradually reduce the disparity.

The cerebral hemispheres in the early second trimester are thin-walled structures with a relatively large ventricular cavity and a smooth external surface. The Sylvian Fissure on the lateral aspect of the brain and the calcarine and cingulate sulci on the medial aspect develop by 18 weeks and the central sulcus by 20 weeks. All the major primary sulci can be recognized by 28 weeks. Development of secondary sulci occurs progressively throughout the remainder of intrauterine life.(2)

The important points to recognize in this structural sequence are the early development of the basal nuclei, midbrain and brain stem and the late but dramatic growth within the cerebrum and cerebellum. The relative sizes and growth activities within different regions of the brain thus change continuously throughout fetal life.

B. Sequence of cell proliferation and migration

Neurons develop by mitotic activity within the ventricular zone of the cerebrum and migrate out along radially-orientated glial fibres to reach their appointed positions within the cortical plate.(3) The cortical plate is first recognizable during the eighth post-ovulatory week. The layered pattern of the cortex is built up by groups of neurons which successively take up positions external to those that have migrated earlier.

The maximum rate of neuronal proliferation within the cerebrum has been shown to occur at 12-18 post-conceptual weeks as the cortical layers are established.(4) After this initial phase of neuronal proliferation and migration, further growth within the cerebral hemispheres is associated with proliferation of cells of the subependymal germinal matrix beneath the walls of the lateral ventricle and migration of these cells into the widening cerebral hemisphere. Most of the subependymal cells are probably destined to form the supporting glial elements.(5) Throughout the latter part of pregnancy, the axons and dendritic spines and processes of the cortical neurons are progressively elaborated, and myelination commences.(6)

C. Sequence of vessel development

The initial blood supply during development consists of a surface capillary network. Penetrating vessels develop to provide a rich supply to the brain stem, midbrain, thalamus and basal ganglia. During the period when there is an actively proliferating subependymal cell plate, branches from a series of major arteries feed into this zone.(7) As the subependymal cell plate disappears, the vascular bed is remodelled. In the last third of pregnancy as the connections of the cortical neurons increase in number and myelination commences, there is progressive increase in complexity of the vascular bed within the cortex and white matter of the cerebral hemisphere. The balance of circulation shifts gradually from a central to a cortical and white matter orientation.(8)

D. Sequence of functional development

Function develops early in the Central Nervous System (CNS), probably by the setting up of rythmical discharges as an essential component of a developing central network of nerve cells.(9) This is associated with a recognizable sequence of appearances of neurotransmitter systems in which catecholamine transmitter systems develop before Gamma Amino Butyric Acid (GABA) and cholinergic systems.(10) Initial spontaneous movements develop by six weeks post-conceptual age, spontaneous electrical activity can be detected in the pons from the 70th day and localized face and hand movements as well as breathing movements can be recognized by 12 weeks. From 20 weeks the fetus swallows amniotic fluid. Older fetuses can regulate their posture and react to maternally-induced changes in position.(9)

Organization of Brain Development

The brain is involved in central control of many important aspects of structural and functional development in other organs. Postnatal survival of the individual is totally dependent on functional integration between the CNS and other body systems. An intriguing question is how the structural and functional development of such a central control organ as the brain is itself controlled.

Two extreme answers might be suggested:

A. Both structural and functional brain development might be totally and rigidly pre-programmed.

B. Functional and some of structural brain development might only occur in response to demands from peripheral organs.

From what has been mentioned above in relation to the development of the layered patterns of the cortex, it is clear that much of the basic structural patterns of the brain must be pre-programmed. There is also good evidence that many of the initial specific axonal connections are similarly predetermined. It has been recognized since the work of Cajal that axonal growth is associated with the development of expansions of the axonal tip, known as growth cones, from which membranous and filamentous cytoplasmic extensions are extruded to allow growth in any appropriate direction. The work of Sperry indicated that most neurons develop specific chemical labels available within the growth cone which match similar or complementary labels on the surface of target neurons.(11) In addition, cells from similar regions of the brain selectively adhere to each other, due to the presence of large molecular weight surface ligands.

However, if all CNS development were rigidly programmed according to such mechanisms, then the brain would have no reserve capacity and would be highly vulnerable to any type of damage inflicted during fetal life. In fact, the CNS commences by considerable overproduction of cells and much of the remodelling during development results from the programmed death of redundant cells. In specific regions of vertebrate brains, 15-85% of the neuron population is eliminated in this way. Failure of the neuron to make synaptic connections may be a major factor leading to cell death. In addition, many areas of the CNS initially develop an excessive number of synaptic connections which are later reduced by a process of functional competition. Thus although the initial connections within the CNS may be precisely determined genetically and initial functional activities are an inate property of the developing tissue, sensory input during development can significantly modify the ultimate form of the nervous pathways. This has been verified experimentally by procedures such as studying the effect on the visual cortex of suturing one eye closed in the newborn macaque(12) or the effect on development of the sensory cortex in mice following selective removal of vibrissae at birth.(13) In both experiments, impaired development of cortical cells deprived of their input stimuli has been accompanied by compensatory hypertrophy of adjacent cortical areas not so deprived.

The brain development model on which we have to consider the effects of adverse influences has therefore two distinct phases. There is an initial well-programmed development of structural connections and initiation of functional activities, but the final pathways are determined by functional competition and sensory feedback mechanisms with elimination of excessive cells and connections.

Types of Adverse Influences Affecting Brain Development

A. Impairment in organogenesis

Because the neural tube closes by about 26 days after conception, the influences causing neural tube defects operate very early, well before development of the placental circulation, for instance. The association of vitamin deficiencies with neural tube defects is not surprising as these could be expected to influence the whole milieu in which the embryo develops.(14) Many other defects of organogenesis involving the CNS have a genetic or chromosomal basis.

The effects of extrinsic teratogenic agents on the human fetal brain may be serious, but do not result in dramatic malformations. Rubella, for instance, causes cataracts, microphthalmia, severe hearing defects and, rarely, microcephaly and mental retardation. From the time sequence of brain development, the most severe effects on cerebral formation could be expected during the time when the cerebrum is growing most actively, that is, at the 10-18 week stage. There is evidence this is indeed so for radiation,(15) although it is contrary to the traditional views of the first trimester as the most vulnerable period.

B. Metabolic defects

The influence of genetically-determined metabolic defects on the developing brain may be subtle and not readily recognizable at a structural level. Indeed, there may be no apparent structural defect in many forms of congenital mental defect, whether the metabolic basis is known or not. In some instances, abnormality is recognizable in the form of abnormal storage material or progressive destruction of formed structural components such as myelin. Some of the most disastrous functional states are, however, associated with minimal evidence of structural disorder.

C. Acute injury during the fetal period

As hinted earlier, there has been confusion over abnormalities of the brain due to interference with normal differentiation and growth and those that result from destructive processes. Porencephaly, encephalomalacias and hydranencephaly represent the end stage of destructive brain lesions which have developed in utero. Such damage may result from an infective process due to cytomegalovirus or toxoplasmosis or an interference with cerebral blood supply. I have recognized several instances where acute anoxic-ischemic damage has occurred in utero as evidenced by dead and encrusted neurons in the basal nuclei and brain stem and the presence of a reactive gliosis. The infants in question died at birth due to damage to the respiratory centre. Cases with less severe damage might well survive, and be thought to have some genetic form of cerebral palsy.

Damage to cerebral circulation is a likely result of any severe hypoxic episode in utero as the fetal brain represents some 12% of total body mass in contrast to the 2% represented by the adult brain. Large geographic areas of the brain will be damaged by regional failure of cerebral perfusion. Situations in which such damage may be anticipated include episodes of acute failure of placental circulation as may occur in some cases of pre-eclampsia with placental infarction or antepartum haemorrhage.

D. Intrapartum damage to the brain

During the process of birth, the brain is susceptible both to direct trauma and to anoxic-ischemic damage. Traditionally, intracranial trauma has been recognized in cases where cranial distortion during birth has resulted in tears of the tentorium or vein of Galen with massive subdural haemorrhage from torn venous sinuses. The quantity of bleeding may not be the most important event as cranial distortion, which is sufficient to tear the tentorium, will of itself impair cerebral circulation and cause shock.(16) A similar effect may result from a pure anoxic-ischemic stress. An important form of intrapartum brain damage is anoxic-ischemic injury to the thalamus, basal ganglia and brain stem.(17) This results from severe shock such as that due to umbilical cord occlusion and will be followed in survivors by development of athetoid cerebral palsy. In such cases the cerebral cortex may be relatively spared. Variations in pattern of damage in survivors of intrapartum asphyxia are probably related to the variable sequence of changes in blood gases and blood pressure or patterns of regional impairment in perfusion of the brain.

E. Neonatal brain damage

The most important forms of cerebral damage in both preterm and term infants are haemorrhagic and ischemic lesions. Haemorrhagic lesions in particular are now known to develop in 40-50% of all newborn infants weighing 1500g or less at birth. These forms of damage are considered in more detail in my second paper, Intraventricular Haemorrhage and Hypoxic Brain Injury.

Metabolic injury may be inflicted on the newborn brain. The most frequent forms of such damage are bilirubin encephalopathy and hypoglycemia. The bilirubin staining of kernicterus presents a similar pattern to that of damage resulting from cardiac arrest, that is, staining of nuclei of the thalamus, basal ganglia, midbrain and brain stem. This may perhaps reflect the ease with which these metabolically-active regions of the brain can be damaged during development. The pathology of neonatal hypoglycemia is rather unclear as it is seldom seen in an untreated form and, if fatal, is likely to be associated with severe anoxic-ischemic damage.

F. Influence of undernutrition on brain growth and development

The influence of undernutrition on human brain development is not easy to establish. Conditions involving chronic maternal undernutrition do not represent a pure situation as there are likely to be additional factors, such as specific vitamin deficiencies, maternal anaemia, impaired maternal growth and poor uterine growth which may account for any adverse influences on fetal brain development. The most pure form of fetal malnutrition is perhaps represented by the infant of the well nourished mother who develops pre-eclampsia with chronic reduction of utero-placental blood flow. The brain of such a growth-retarded infant at term is large relative to the bodyweight, but of low weight for the gestational age. Structural maturity appears normal, but there is clearly some constraint on cell proliferation. I am not aware that detailed studies have yet been made on relative proportions of different cell types and degree of synaptic development in such cases.

Some of these infants do prove retarded if they survive. However, it cannot be assumed that this is a direct effect of undernutrition as the utero-placental ischemia may result in anoxic-ischemic episodes in utero or during labour and the infant may have suffered the additional effects of hypoglycemia or other metabolic problems.

The effects of postnatal undernutrition have been extensively studied in animals, and deficits in total cell numbers and proportions of different cell types established.(18) The study of the effects of human infant malnutrition on brain development are as difficult to interpret as those of prenatal undernutrition, as there are many confounding factors.

Interactions Between Adverse Factors and Processes of Normal Brain Development

The eventual effect of any adverse factor on the developing brain will depend on the nature of its interaction with the normal processes of brain growth.

Prevention of some important time-related event in organogenesis, such as closure of the neural tube, is a once only event and the results are inevitable. Impairment in proliferation of cells of the cerebral cortex during the early second trimester or disorganization of patterns of migration will clearly disturb some of the primary programming of cell connections. If such an influence does not exceed the reserve capacity of the brain to allow for failure of axonal connections, there need be no adverse effect on eventual CNS function. Indeed, we do not know to what extent the normal brain at birth is oversupplied with cells. A further possibility is that a decrease in neuronal population may be compensated for by an increase in synaptic connections or glial development. Thus, it may not be entirely surprising that a general growth-retarding stress, such as rubella infection, does not always cause microcephaly or severe mental retardation even if it does act when the cerebral neurons are most actively proliferating at 10-18 weeks.

Some of the most striking forms of perinatal brain damage are the common haemorrhagic and ischemic lesions. The frequency of such lesions revealed by computerized tomography and real-time ultrasound examinations in life, has surprised paediatricians who have been unable to detect clinical evidence of CNS abnormality in many of the infants. In visualizing how much lesions may interact with normal developmental processes, it is important to consider the time at which the damage occurred. A localized area of haemorrhage or necrosis in the periventricular region of the brain of a 28-week gestation infant involves a mass of tissue in which synaptic connections are rudimentary, myelination has not commenced and many of the cells (e.g., subependymal cell plate) may be destined to die. In such a case, there may be a very considerable capacity for remodelling during later development. Any remodelling will, however, be limited by the extent to which the vital permanent structures already developed (e.g., basal nuclei) are damaged, and the volume of brain destroyed in relation to the size of a particular field of developing cells. The same principles would apply to similar damage developing near term. However, the increased differentiation of permanent neural pathways will place narrower constraints on the extent of remodelling possible in the more mature infant brain.

How can we exert favourable influences on brain development and mitigate the effects of unfavourable ones? From a purely negative point of view, we can eliminate damaged fetuses by a policy of selective abortion following antenatal detection. We may attempt to improve the milieu for neural tube development by vitamin supplementation before conception. We can attempt to avoid or limit unfavourable influences such as those which lead to haemorrhagic and ischemic lesions. It is, however, in some ways more interesting to speculate whether it may be possible to influence remodelling and thus to mitigate the effects of cerebral damage after it has occurred. The aspect of development we would most like to influence is intellect, although it would be nice to be able to influence motor, speech, visual and hearing abilities too. There is no quantitative assessment of how effective this might be in human infants. It is, however, well established that young rats reared in a stimulating environment in a large group with interesting play objects which are changed daily, have increased brain weight and cell population in comparison with their non-stimulated litter mates, although they do not gain so much body weight. Such stimulation can compensate for many of the effects of nutritional impairment.(19) Winnick and his colleagues have provided evidence that this may be true also for malnourished infants. This type of observation in conjunction with the data on special sense organ development mentioned earlier, supports the concept that sensory input is of major importance for CNS development.

The vogue for establishment of early mother-infant bonding with the various forms of infant stimulation that such rituals involve may be regarded by some clinicians as a passing fad. The developmental neurobiologist would question whether the forms of sensory stimulation involved might aid in determining the ultimate structure of important neural pathways. This is perhaps an area which merits further investigation. It may well be that is not possible to improve on a 'normal' level of stimulation and that 'stimulated' experimental rats are in fact normal animals. Many paediatricians might be unhappy if the enthusiastic methods of some cerebral palsy treatment regimens were to be introduced to the neonatal intensive care unit. However, the isolette of the neonatal intensive care unit is perhaps akin to the solitary cage of the deprived rat, and there may be ways in which both intellectual and motor development could be encouraged by appropriate forms of sensory input.

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MATERNAL SMOKING DURING PREGNANCY AND CHILD DEVELOPMENT: A REVIEW, AND THE METHODOLOGY FOR A NEW STUDY OF OVER 10,000 REPRESENTATIVE FIVE YEAR OLDS IN GREAT BRITAIN

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The effect of maternal smoking during pregnancy on fetal growth and immediate survival is profound; the effect on later development is unclear. The longitudinal study of the 1970 British national birth cohort, initially called British Births, and now Child Health and Education in the Seventies (CHES), has offered a unique opportunity to better clarify the relationship of maternal smoking to early child development. By using the extensive data on this cohort of over 16,000 children born in Great Britain, April 5-11, 1970, collected at birth and age five years (and soon, at age ten years), we aim to judge whether there are residual effects on the child of maternal smoking during pregnancy.

We must therefore distinguish whether relationships between maternal smoking during pregnancy and subsequent child development are direct, and therefore possibly causal, or indirect. The data available afford a particularly strong test of this question: we can compare outcomes for children whose mothers smoked throughout pregnancy with those who had smoked at the same level, but had stopped; and we can control for social differences between smokers and non-smokers, by use of a comprehensive social analysis, including measures of social drift over time, differential use of pre-school educational and other services, and a rich array of other social and behavioural variables, much beyond the traditional index: father's occupational status.

There is good evidence that the toxic effects of smoking on the fetus are more intense among those of low social status (we have hypothesized that such effects may be mediated by differences in maternal nutrition). We are therefore studying whether there are effects of maternal smoking on later child development also contingent on social status, maternal (and childhood) nutritional status, and interrelated patterns of these variables.

While maternal cigarette smoking during pregnancy is profoundly related to the growth and survival of the fetus, the nature and extent of effects on the later development of the child are not at all clearly understood. Past attention to this problem has been sparse, and the issues are beset with inherent difficulties that are unlikely to yield easily to even the clearest and most elegant research strategies. Nevertheless, the consequences of maternal smoking for the child have been posited to be profound, and the potential importance of this problem has justified our intensive and extensive research.

Review of Past Work

Davie, Butler and Goldstein(1) and Butler and Goldstein(2) reported on the relationship of maternal smoking during pregnancy on children aged seven years and eleven years, who were studied in the longitudinal follow-up of all births in Britain during one week in March, 1958: The National Child Development Study (NCDS). They reported both on relationships with and without adjustment for social class, birth order, mother's height, number of younger siblings, mother's age, child's sex, but also birthweight (which we now consider to be analytic over-control: see below). The reported results are summarized in Table 1:

Maternal Smoking
(at the 4th Month of Pregnancy)

	less than 10 cigarettes/day					10+ cigarettes/day				
	Age 7			Age 11		Age 7			Age 11	
	raw	Adj. w/o birth-weight	Adj. with birth-weight	raw	Adj. with birth-weight	raw	Adj. w/o birth-weight	Adj. with birth-weight	raw	Adj. with birth-weight
Height (cm)	0.9	0.63		.95	.55	1.3	0.99	0.6	1.5	1.0
General ability (months)				7.8	4.5				7.8	3.3
Reading Comprehension (months)		1.5(1)	1.0	7.5	3.0		4.8(1)	3.9	9.1	3.6
Mathematics ability (months)				7.2	4.6				8.5	5.0

Developmental Delay, in months (for height in cm) at ages seven and eleven, associated with maternal smoking during pregnancy, National Child Development Study, raw values, and adjusted for social class, birth order, mother's height, number of younger siblings, mother's age, child's sex, with and without adjustment for birthweight.

(1) calculated from authors (4) data, assuming 100 gm decreased birthweight for those smoking less than 10 cigs/day, and 200 gms for those smoking 10+ cigs/day.

Table 1

Fogelman(3) has analyzed the relationship between maternal smoking in pregnancy and height, reading test score and mathematics test score, at age 16 for the 1958 birth cohort. With birthweight controlled, there were small differences in reading and mathematics scores of children whose mothers smoked up to 10 cigarettes per day. Male offspring of women who smoked more than 10 cigarettes per day were approximately 0.9 cm shorter than male children of non-smokers. No significant differences were present among females. These effects of maternal smoking during pregnancy are over and above those mediated by birthweight.

Hardy and Mellits(5) did a small matched-pair study (at ages four and seven years) among the Baltimore Collaborative Perinatal Study population. One set of controls was matched for birthweight: since one of the major pathways for the effect of smoking is to decrease birthweight, controlling for birthweight masks any effects of smoking mediated by depressed birthweight. Such over-controlling was also done in the analysis of the NCDS, and in the Vancouver and Newcastle studies (see below). For the 88 pairs, without birthweight control, children of smokers were 1.0 cm shorter at age seven years (not significant), about the same magnitude as the NCDS. Children of smokers were significantly worse in spelling section of the Wide Range Achievement Test (W.R.A.T.); there were no other significant differences on a battery of psychological tests.

Denson(6) reported that mothers of methylphenidate-sensitive hyperkinetic children smoked two or three times as many cigarettes in pregnancy as mothers of dyslexic or control children.

Dunn(7) demonstrated fairly severe and consistent developmental retardation on a wide range of outcomes at age six and one half years associated with maternal smoking in pregnancy. Again, they also controlled for birthweight (They actually did their analyses within birthweight strata). They thus understated the relationship of developmental delay and maternal cigarette smoking, since they were, in effect, reporting effects present over and above those mediated by effects on birthweight.

Kolvin(8) (personal communication) found a strong association between maternal smoking and developmental delay at ages five to seven years. After controlling for birthweight, differences became minimal. In spite of their conclusions, their study suggests strong associations of maternal smoking with depressed child development.

The weight of available evidence thus suggests fairly strong association of maternal smoking in pregnancy and childhood developmental delay, on a wide variety of behavioural indices and stature.

There must remain, however, deep reservation over whether the associations are causal in nature. The problem is nicely illustrated by Douglas'(9) retraction of an earlier report associating low birthweight with subsequent depressed mental ability and school achievement. After restudy of the children and normal birthweight controls, he wrote:

> "In a national study of the mental ability and primary school progress of 'premature' children a number of striking handicaps were found, which were later shown to be of environmental origin rather than result of low birth weight per se....These differences are largely explained by the fact that premature birth is not only associated with poor living conditions but also, at each social level, with low standards of maternal care and lack of educational interest."

The matching procedure failed to fully match on social status. In the body of the paper, Douglas stated:

> "As this survey progressed it became increasingly clear that, in spite of the careful matching of the premature and control pairs at the beginning of the survey for father's occupations, there were, in fact, marked social differences between them which have grown larger with time. The fathers of the premature children were more likely to move during the survey period to less favourable occupations than the fathers of the controls, and were less likely to improve their occupational status or move into self-employment. Moreover, periods of unemployment were more than twice as frequent among them -- 6.5% were unemployed at one or more occasions as compared with 3.2% of the fathers of the controls."

Smokers are known to be different in behaviour, personality, and social status from non-smokers.(10,11,12,13,14,15) Thus, social and behavioural differences between parents who are smokers and non-smokers must be carefully determined, and the effect of these differences taken into account, before the effects of maternal smoking on child development can be sorted out with any certainty.

Not only must social factors be controlled, but effects must be explored, contingent on social class. There is strong evidence that the effects of maternal smoking on fetal growth and survival are far worse among lower class women,(16,17) and the possibility of effects of maternal smoking on later child development contingent on lower social status must therefore be considered.

THE CHES STUDY

Over 90% of all children born in the United Kingdom, April 5-11, 1970, were studied as part of a survey entitled "British Births". (16,882 of them were born in England, Scotland and Wales.) A questionnaire was completed by midwives (with the help of obstetricians and paediatricians) from maternity records and interviews with mothers. The findings on the first week of life(18,19) and further analyses on complications of pregnancy and labour have been published.(20) This material, all on computer tape, has been linked with the later progress of the surviving children at age five years. The questions asked at confinement on smoking were:

a) Does the mother smoke now? Yes, No, or Not Known
b) If NO, did she ever smoke? Yes, No, or Not Known
c) If YES, how long ago did she stop? ______ Years _______Months
d) How much does/did she smoke? Cigarettes per day, _____1-4, ____5-15, ____ 15-24, _____ 25 or more; _____or Not Known.
e) Has she smoked during this pregnancy? Yes, No, or Not Known

Babies of smokers weighed 170 grams less than those of non-smokers, with an average of 0.1 weeks reduction to gestational maturity, results typical of past observation.

Extensive other data were gathered. From a detailed description of father's occupation, social class was coded. Other social data included mother's occupation, marital status, place of birth (of mother, father, and mother's mother) and age at which education was completed (both mother and father). Sociobehavioral data included date of first prenatal visit, and number of visits. Medical information on mother and infant was detailed.

A subsample of 1,000 study children living in the South West Region of England and South Wales at the age of three and one half years took part in a special follow-up study. Many of the questions and techniques successfully used in this regional study provided reliability checks on some of the data gathered in the national study at age five years, called Child Health and Education in the Seventies (CHES). Techniques were developed to assess yearly attendance at playgroups, nursery schools, early utilization of child health services and details of mother's earlier employment.

The CHES study involved an investigation of the fate, health, development and preschool education of the national cohort at age five years. Two percent had died before five years, leaving 16,306 children eligible for follow-up. Tracing was carried out in 1974 through the National Health Service Central Register and with the cooperation of almost every Area Health Authority in England, Scotland, and Wales.

The aim was to document the total early experience of these five-year-old children inside and outside the home, and to relate perinatal and early experience to general development at statutory school entry age (five years in England). Many factors thought to have implications for early development were documented and in particular the effects, within differing social, family and physical environment, of early childhood separation from parents, the duration and causes of early hospitalization, the nature and duration of accidental injury and poisoning, and of physical and mental handicaps.

Interviewing and testing on 13,135 (80.6%) five-year-old children was carried out by Health Visitors in 1975. The questionnaire included one given to the parents by the health visitor, one completed by the mother, and tests of development given to the child. In order to increase the returns, the whole sample was retraced in 1976 from school records through Local Education Authorities. Health visitors then completed a shortened version of the parental home interview questionnaire on a further 1,965 of the study children (12.1%), none of whom had taken part in the 1975 study. Thus, information at five or six years is available on 92.7% of all the children, the remainder comprising incomplete information (0.9%), refusals (3.8%), and untraced (2.6%). Coding, punching and data-file preparation began in 1976 and by the end of 1977, 228,040 cards had been punched and put onto magnetic tape (18,243,200 bytes).

During 1976 and 1977, validation was performed on certain parts of the material. On children living in the county of Avon at the time of the five year study, details of admissions and out-patient attendances were validated from hospital records. The reliability of neighborhood ratings made by the Health Visitors has been assessed within Avon. The educational tests used at five years were repeated in Hertfordshire by Speech Therapists and Health Visitors, to confirm test-retest and inter-observer reliability. National validation has been done from hospital and GP records on the children with convulsions, fits and seizures, and also on approximately 1,000 children diagnosed at five years to have handicaps and disability.

Data collected included the following:

A) Home interview with child and mother: Structured information was gathered by a health visitor on:

1) parental smoking habits;
2) family composition;
3) the child's past medical history and current health;
4) health of other members of family;
5) the child's experience within the home, especially television and reading practices;
6) child's experience outside the home;
7) parental education and occupation; and other social information;
8) family housing, living conditions and social environment.

B) Estimates of Child Behaviour, Maternal Anxiety and Parental Attitudes included:

1) the Rutter B-Scale child behavioural questionnaire(21)
2) a maternal "Malaise Inventory" developed by Rutter et al.(22) from the Cornell Medical Index, to detect problems in the mother's own health; and
3) a set of 50 attitudinal questions for self-completion by parents, mainly relating to views on child rearing practices.

C) Details of child's Community Health Records

This was completed from records of health care. It included:

1) the extent, nature, and times of all health contacts and screening procedures;
2) details of all assessments, special tests and hospital admissions or attendances; and
3) details of whether the child had ever been categorized for special education or to be on any at-risk or handicapped register, including actual suspected non-accidental injury.

D) Graded tests of child development

This was a series of tests appropriate to the child's age and carried out by health visitors at the time of the home interview. They comprised:

1) a copying-designs test;
2) Draw-a-Man Test (human figure drawing);
3) English Picture Vocabulary Test;
4) the Complete-a-Profile Test;
5) Graded Word-Reading Test (Schonell);
6) measurement of height and head circumference.

The questions on smoking (for both mother and father) were:

1) Do either N's mother or father smoke at all at present? (Cigarette smoking is defined as smoking an average of one or more cigarettes a day.) No, is a non-smoker; Yes, smokes cigarettes; smokes pipe or cigars; Not known if smokes; If smokes cigarettes, how many are smoked per day on average?

2) Irrespective of whether or not N's mother or father smoke at present, for how many years since N's birth have they smoked cigarettes, If at all? During the period since N's birth - Smoked all the time _____; Smoked for more than three years _____; Smoked for between one and three years _____; Smoked for less than one year _____; Smoked but not know for how long _____; Not known if smoked at all _____.

The history of parental smoking between birth and five years was as follows:

	Mother	Father
Cigarettes, throughout	32.8%	42.1%
Cigarettes, some of the time	15.2%	13.2%
Cigars, pipes and other	10.9%	13.2%
Non-smokers	41.1%	28.1%

Social class, classified by father's occupational status, has shown that 32.8% of the cohort are non-manual and 60.6% manual.(23) The type of neighbourhood in which the study child lived has been rated, and employed as an indicator of the child's probable priority in health surveillance and educational provision. A classification has been devised whereby the type of neighbourhood in which the child lived could be divided into four mainly urban groups: poor (7.6%), average (48.1%), well-to-do (22.5%) and inner urban (3.2%) with a fifth category for rural neighbourhood (18.3%).

The five tests used to assess the child's mental aptitude and achievement were presented in the following order:

1) Copying Designs Test
2) Human Figure Drawing
3) English Picture Vocabulary Test
4) Profile Test
5) Schonell Reading Test

1) Copying Designs Test

Design copying is a feature of many intelligence tests and may be regarded as a measure of general ability. The items used are taken from established I.Q. tests such as the Stanford-Binet and Wechsler scales and similar tests have been used in earlier cohort studies of child development.(1,22)

Eight different designs were presented, and the child was required to make two attempts at each design. The designs have been scored using the same criteria as Rutter and his colleagues devised for the Isle of Wight studies, which conform to standard procedures.

2) Human Figure Drawing

In this test, the child was invited to "make a picture of a man or lady" on a blank page in the Test Booklet. A second drawing was then requested of opposite sex to the first on a new page.

Children's representations of the human figure have long been recognized as following a definite developmental sequence.(24) Although it has been suggested that elicited drawings do not provide true indications of a child's cognitive level and that samples of spontaneous art are more valid for this purpose,(25) the test developed by Florence Goodenough in 1926 was proved to be reasonably predictive of other psychological measures using standard forms of test.(24) For example, for children aged about five years, correlations of the Goodenough-Harris scale Draw-a-Man Test with Stanford Binet scores were found to be 0.45 (N = 90) for mental age and 0.41 (N = 90) for I.Q. (op. cit., pp. 96-97).

Validity and reliability checks produced product moment correlations between Goodenough-Harris and Koppitz scores of r = 0.72 for five-year-old boys and r = 0.70 for five-year-old girls. The Koppitz scale and scoring method produce correlations with Stanford-Binet I.Q. scores of 0.62 and with WISC Full Scale I.Q. scores of 0.60 for children aged six and seven years.(26,27)

3) English Picture Vocabulary Test (E.P.V.T.)

The E.P.V.T. is the British version of the Peabody Picture Vocabulary Test in which the child is asked to indicate the one picture of four which corresponds to a word spoken by the tester. A series of 56 sets of four pictures is presented with the test words increasing in difficulty throughout. The E.P.V.T. is essentially a word comprehension test, and it was the only established test available containing an element of language skill which could also be feasibly implemented in a study of this size.

4) Profile Test

This test was developed by Alex F. Kalverboer, Department of Clinical Psychology, University Hospital, Groningen, The Netherlands, and is based on Piaget's theory of spatial-constructive development in children.(28)

Children are presented with a page containing an outline of a head in profile and are invited to "Draw everything that is missing on it." The average child in the four- to five-year age group typically "completes" the profile by adding two eyes, a nose, a mouth and sometimes two ears in typical full-face arrangement. After about five years of age there is a gradual transition from full-face to profile completions.

5) Schonell Reading Test

A minority of children in this sample were expected to have begun to learn to read by the age of five years. The Schonell Reading Test consists of a card containing 50 words printed in large type. In this test, children must not only read the words on the card, but also articulate them. In view of this, the results are likely to underestimate the proportion of children in the sample who can read a few words. In addition to the test itself, mothers are asked to say if they think their children can read letters, words or simple sentences. The data on reading ability has been used only as a rough indication of the small proportion of children who are beginning to read at this young age. Preliminary results on a subsample of children have shown that about a third of five year olds attempted the test and achieved a score. A further tenth attempted the test but did not achieve a score, and the remainder (56%) did not attempt the test.

Child's Measurements

In addition to the cognitive tests, the child's height and head circumference were measured. These measures have been obtained for comparison of norms obtained in the 1946 and 1958 cohort studies(1,9,29) and to investigate further the relationship between social and environmental factors and physical development.

BEHAVIOURAL AND PSYCHIATRIC MEASURES

Behaviour Rating Scale

A child's ability to adhere to generally-expected behavioral norms is of some importance at the age of school entry. Fidgety, clumsy or other generally misdirected behaviour is often associated by teachers with poor concentration and an expectation of lower than average school performance(30). It is thought that such expectations themselves contribute to a reduction in a child's likelihood of achieving his/her full educational potential.(31) Given the relationship between childhood hyperactivity and maternal smoking,(6) these issues are particularly important to this study.

The behavioural scale used is that originally developed by studies in the Isle of Wight. A similar scale was also used in the 1958 cohort (National Child Development Study).(1)

The scale successfully identified 70.8% (N = 120) of children attending psychiatric clinics for emotional or behavioural disorders, and only 11.6% (N = 198) of children randomly selected from the general population.(21) These results suggest that mothers and fathers are usually in agreement over the behavioural characteristics of their children and their assessment remains reasonably stable using this instrument. The scale also discriminates at a satisfactory level between a group of children with known behavioural or emotional problems and a sample from the general population.

Maternal "Malaise" Inventory

To assess maternal mental state, use was made of a 24 item scale based in part on the Cornell Medical Index and modified for use in surveys by Rutter, Graham, and Yule,(21) who found that three-fifths of mothers clinically diagnosed as having psychiatric disorders were identified using the inventory, compared with only one in twenty of mothers without psychiatric disorder.

Maternal Attitudes

An array of questions was asked of the mother, concerning attitudes to child-rearing, discipline, growth and development, and the importance of work for women and its effect on children.

METHODS

We faced the challenge to make optimal use of this extensive evaluation of both the child's development and the milieu in which he or she spent the first five years of life. Prior studies of maternal smoking during pregnancy and subsequent child development had, on one hand, overcontrolled (by removing the effects of cigarette smoking mediated by changes in birthweight, when these might well be the predominant associations with smoking), and also undercontrolled for differences between characteristics of families of smokers and non-smokers.

We restricted our analysis to singleton births, in families of British or European origin in which the child resided with the biologic mother, and where English was the principal language spoken, and to children with complete information on dependent variables (incomplete information might exist on one or another of the independent variables). Thus, we were left with 10,123 subjects of the 13,135 who had participated in the survey at age five years.

In order to reduce the number of variables in analyses, we first subjected each of the arrays of control variables (as well as the Rutter Child Behavior Scale) to varimax rotated factor analysis. After judging the best solution, factors were used to cluster items, within each variable set. Appendix I includes the clusters of items around issues of the social conditions of the family, the clusters for the Cornell Medical Index items and the three clusters from the Maternal Attitude Questionnaire. Appendix II gives the clusters of items derived from the child behaviour items. The clusters included all items that loaded 0.3 or higher on the factor, and the cluster score for any individual consisted of the average standardized score on all items included in each cluster. This allowed us to score individuals even if all but one item was missing. In addition, we used several individual items for control purposes: mother's height (particularly important in assessing the relationship of smoking to the child's height and head circumference); the mother's history of whether she was a late reader, poor reader, had convulsions, was a bed-wetter, or was a late speaker, and the child's preschool experience (whether the child had been to a private preschool, public preschool, had early entry to regular school, or none of these).

Hierarchical multiple regression analysis was performed to control for differences in distribution between smokers and non-smokers for these various issues and also to control for amount smoked. We controlled for the age and sex of the children, then for social factors, maternal height, developmental history, attitudes, malaise inventory, and then on the child's pre-school experience. The only change in this sequence was for dependent variables, the child's height and head circumference, where maternal height preceded social factors. Since height and social status are strongly related, the logic of this decision is based on the premise that the contribution of height to physical growth was a more direct one than to the mental measures.

RESULTS

Extensive preliminary results were presented at the conference, but publication of these results must, for the moment, be deferred. We will attempt to prepare as an addendum to next year's conference proceedings to summarize the results of age five years, and possibly, make a preliminary statement about the reexamination of these children at age ten years. The results remain complex, but in summary, most effects are markedly attenuated by control for social status, although they persist, albeit at lower magnitude.

APPENDIX I

Component Items of Clusters Used for Controlling

"SES"

Mother's education
Father's education
Father's social class
Tenure of accommodation
Owns telephone
Economic level of neighbourhood

Number of older children
Maternal age
Number of subsequent children
Number of household moves since birth

OTHER SOCIAL FACTORS

Number of weeks father unemployed
Owns refrigerator
Owns automobile
Quality of furniture

None, B.&W., colour T.V.
Relationship with neighbours
Bathroom in household
Number of rooms in accommodation
Tidiness of home

Mother employed for:
  Necessity
  Advantage
  Social reasons
  Career
  Family business

Father figure absent
Father away evenings
Father away weekends
Father works shifts

Father's 1970 occupational group

Social class change, 1970-75

MATERNAL DEVELOPMENTAL HISTORY

Late reader
Poor reader
Convulsions
Bedwetting
Late speaker

HS170F1

Appendix I (Continued)

MATERNAL ATTITUDES	PRESCHOOL EXPERIENCE	MATERNAL MALAISE
Child with only his own ideas will not learn	Private preschool	Tired
Playing takes second place to reading	Public preschool	Depressed
Girls should accept marriage, not career	Early entry to regular school	Worried
Hospital routine is upset by visiting		Violent rage
To not let others stand in your way is admirable		Annoyed by people
Children should not talk at table		Easily upset
Children should accept what parents say		Everything gets on nerves
Visiting child in hospital is upsetting		
School freedom lets education standards fall		Indigestion
Child should not talk back		Upset stomach
Child must do things without explanation		
Parents should sort out children's quarrels		Worry about health
Equality O.K., but husband has main say		Suddenly scared
Person should love, respect parents		Scared to be alone
People should be satisfied, not struggle for more		Scared to go out
Giving in to a child spoils him		Heart races
Child cannot understand others' feelings		Sleeping problem
Good child is one not told twice		Wake too early
Child upset in hospital soon gets over it		Poor appetite
Vandalism is due to lack of discipline		
Parents should treat children as equals		Backache
Respect for authority is not important		Headache
Child must be himself		Pains in eyes
Teenagers have different morals than adults		
Mother's place is in the home		
Women need more from life than family		
Mothers need a break from children		
State should open day nursery for working mothers		
Mother can work if child care is O.K.		
Mother who works is not fit to be a mother		
A wife with children has no right to work		
Mother should accept child is too busy to obey		

APPENDIX II

Component Items of Clusters Derived from The Rutter Child Behavioral Questionnaire

Anti-Social Behaviour

Temper tantrums
Destroys belongings
Fights
Irritable
Takes things
Disobedient
Tells lies
Bullies

'Neurotic' Symptoms

Often worried
Solitary
Miserable/tearful
Fearful
Fussy

Sleeping Difficulty

Falling asleep
Night walking
Early morning waking
Nightmares

Restless, Unsettled

Very restless
Unsettled
Squirmy

Enuresis/Encopresis

Bedwetting
Day wetting
Soiling

Physical Discomfort

Headaches
Stomach aches
Biliousness

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Speakers and Moderators

INTRAVENTRICULAR HAEMORRHAGE AND HYPOXIC BRAIN INJURY

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Introduction

The links between haemorrhagic and ischemic lesions in the developing brain are gradually becoming apparent as pathological studies are co-ordinated with investigations of developmental vascular anatomy and cerebral circulatory physiology. The new imaging techniques offer great potential benefits for diagnosing and monitoring these lesions but the full potential of such methods can only be achieved if they are used as adjuncts to clinical, physiological and anatomical studies.

In this presentation, discussion will be restricted largely to intraventricular haemorrhage and hypoxic injury in the preterm infant as time and space will not permit consideration of the whole range of hypoxic and ischemic lesions which may affect the infant at term.

Intraventricular Haemorrhage

A) Site of Origin

As this condition comprises the presence of free blood or blood clot within the ventricular system there are a variety of ways in which it can develop. Origins of bleeding suggested in the literature have included the choroid plexus, the terminal vein or its tributaries and areas of venous infarction.(1) To some extent variations in suggested origin of the bleeding may in the past have been related to variations in the population of infants seen at post mortem examination.

In recent years the ability to detect bleeding in life using computerized tomography or ultrasound scanning techniques has given added impetus to the study of intraventricular haemorrhage (IVH), particularly in very low birthweight (VLBW) infants treated in neonatal intensive care units. In this group of cases the bleeding most often commences as a haematoma within the subependymal matrix overlying the head or body of the caudate nucleus with subsequent rupture into the ventricular system. Preoccupation with this form of IVH should not cause it to be forgotten that bleeding into the ventricles may occur from sites other than this, even in VLBW infants. It is also important to remember that IVH is quite common in relatively mature infants. In these, the site of origin is perhaps more likely to be the choroid plexus as subependymal haemorrhage can often not be detected with scanning techniques. Atypical sites of origin of IVH which may sometimes be recognized in both preterm and mature infants include areas of infarction in the cortical white matter or basal ganglia.

Injection studies performed on brains of small preterm infants indicated that the common subependymal haemorrhage (SEH) represented a breakdown of the capillary bed or rupture of capillary vein junctions within the subependymal matrix.(2) It was noted that the arteries supplying the subependymal matrix were relatively large in the infant of 24 - 30 weeks gestation, implying that a considerable proportion of total cerebral blood flow must perfuse this tissue in the preterm infant. These purely anatomical observations indicated that factors influencing cerebral blood flow in the VLBW infant might be of central importance in the pathogenesis of this particular lesion.

B) Pathogenesis of SEH/IVH in the Small Preterm Infant

On the basis of our anatomical studies, we originally suggested that disruption of the microcirculation within the subependymal matrix might result from transient rises of arterial pressure associated with apnoeic attacks in preterm infants. This effect would be accentuated by the cerebral vasodilatation caused by the hypercapnia and hypoxia of the respiratory distress syndrome (RDS). The soft cranium of the preterm infant will allow increases in arterial pressure to be accompanied by more pronounced changes in transmural pressure than would be the case with a rigid skull. Studies on cerebral blood flow (CBF) using the Xe^{133} method have documented a low CBF in asphyxiated preterm infants and an apparent relation between CBF and blood pressure in such infants.(3) On the basis of these observations it was suggested that asphyxia caused loss of autoregulation of CBF in preterm infants and that overperfusion resulted when the blood pressure subsequently returned to normal or high levels. It is not yet clear whether the small preterm infant has the ability to autoregulate CBF in the classical way, that is, to maintain a relatively constant CBF over a wide range of blood pressure. Even in adult animals and man autoregulation is abolished by hypercapnia or hypoxia. Thus the lack of autoregulation in infants with asphyxia or RDS is no more than the expected result of the blood gas changes. Low CBF associated with a fall of blood pressure during asphyxia may cause an impairment in the ability to autoregulate, which persists after the blood gas tensions return to normal, but this remains to be proved. An alternative possibility is that low CBF causes direct ischemic damage to the integrity of the capillary bed within the subependymal matrix. In either event a subsequent rise of blood pressure to normal or increased levels might cause haemorrhage due to disruption of the periventricular capillary bed.

Although it is not yet clear whether one of these sequences may occur more frequently than the other, the major problem expressed by both is poor control of CBF in the sick preterm infant. It seems probable that there may be quite rapid major swings between states of hypo- and hyperperfusion of the brain in these infants.

Information From Real-Time Ultrasound Studies

The ability to recognize IVH in life and to study its evolution with serial ultrasound studies has thrown new light on the natural history of the condition. Most cases of SEH/IVH can be recognized within the first 48 hours of age and large bleeds can sometimes be seen within three to four hours of birth. A very high incidence of IVH at birth or within the first 12 hours of age has been claimed by one group,(4) although we have not been able to substantiate this in our own material.(5) Bleeds tend to develop earlier and to be of larger size in the least mature infants. In infants admitted to the Hammersmith Hospital NICU, the incidence of bleeds is very high in those below 31 weeks gestational age but falls markedly in those of greater maturity.(M.I. Levene - personal communication) This suggests some change in the subependymal microcirculation at this stage of gestation.

Relatively few bleeds develop after three days of age. This would indicate either that most cases of SEH/IVH develop as a result of some problem or problems directly associated with birth or that there is a rapid change after birth in the pattern of regional CBF or in the structure of the periventricular capillary walls.

Comparison of a series of ultrasound pictures taken in life with the findings at post-mortem examination in infants who die confirms the view that several different pathogenetic sequences may result in SEH/IVH. In some instances, the lesion comprises pure haemorrhage whereas in others an episode of infarction may constitute an important stage in the sequence of events.

In older studies an association was established between RDS in life and the recognition of SEH/IVH at post-mortem examination. Since it is now known that many infants have clinically unrecognized SEH/IVH and a high proportion survive the condition, it is important to confirm the clinical associations in the living population. Several studies have now shown that there is indeed a highly significant correlation between RDS, (and its individual features such as hypercapnia and acidosis), and the development of SEH/IVH as diagnosed by real-time ultrasound scanning.(6)

Although large bleeds may not be accompanied by dramatic clinical symptoms the use of a range of selective neurological tests has shown that certain clinical features are significantly associated with the presence of an SEH/IVH.(7) Impaired visual tracking, a tight popliteal angle and the later development of roving eye movements have been shown to be significantly associated with presence of IVH in infants of 31 weeks gestation or less.

Periventricular Leukomalacia and its Relation to SEH/IVH

As it has now become easy to recognize SEH/IVH by use of scanning techniques in life, there is a tendency to think that this is the only important form of vascular damage to the preterm brain. Periventricular leukomalacia, the main form of hypoxic-ischemic brain damage in the preterm infant, is a frequent concomitant of SEH/IVH and is believed to be an important cause of severe neurological handicap in preterm infants.(8,9) The lesion is a form of ischemic necrosis of the periventricular tissue due to impairment in cerebral circulation, probably resulting from episodes of hypotension developing during apnoeic attacks in small preterm infants. The periventricular region in the preterm infant represents a boundary zone between blood vessels passing in towards the ventricle from the cerebral cortex and those which penetrate up close to the ventricle from the base of the brain and supply the ependyma and adjacent subependymal tissue. In the early phase, periventricular leukomalacia (PVL) is recognized as a slight softening of the brain tissue with early coagulation necrosis. At a later stage there may be 'white spots' due to areas containing groups of fat-laden histiocytes. In an infant who survives for several weeks, there may be gliotic scars interspersed with cystic areas from which the necrotic brain tissue has been removed. As the lesion involves destruction of future white matter it may prevent development of the cortico-spinal motor pathways and result in spastic diplegia or quadriplegia. Destruction of the optic radiation may be expected to interfere with development of visual function.

As has been explained above, episodes of both hypoperfusion and hyperperfusion may be involved in development of SEH/IVH. The frequent co-existence of PVL and SEH/IVH is therefore hardly surprising.

Unfortunately, it is not yet possible to visualize the early stages of ischemic lesions such as PVL with any certainty by use of CT or ultrasound techniques. The later cystic stages of PVL can be recognized by ultrasound particularly if a sector scanner is employed for this purpose.(M.I. Levene - personal communication)

Evolution and Sequelae of SEH/IVH and PVL

Small haemorrhages may resolve with little evidence of abnormality although small cystic lesions in the subependymal matrix can often be recognized on ultrasound scans and are frequently found at necropsy in infants who die of other complications of prematurity. Some degree of ventricular dilatation has been recognized in about 40% of cases of SEH/IVH studied at Hammersmith Hospital.(10) Since real-time ultrasound or CT scanning allows ventricular dilatation to be diagnosed in the absence of a rapidly increasing head circumference there is a tendency to submit the infants to drainage procedures in order to prevent development of overt hydrocephalus. However, at Hammersmith Hospital, where no treatment has been given for an increased ventricular size alone, only 21% of the infants with enlarged ventricles have subsequently developed a rapidly progressive hydrocephalus requiring treatment. The remainder have stabilized or resolved spontaneously. This does not, of course, answer the important question as to whether the increased ventricular volume causes any damage to the brain which might be prevented by sequential tapping.

Follow-up studies have generally shown bad results for infants with large haemorrhages which fill and dilate the ventricles or are associated with extension into the cerebral substance.(11,12) Haemorrhages involving the subependymal matrix only or with ventricular spread without dilatation have in general been found to have a good prognosis for neurological development at a year of age. It is not easy, however, to evaluate these findings fully as any associated ischemic necrosis is unlikely to be visualized by CT or ultrasound scanning methods, until cystic lesions have developed. The quantity of brain destroyed may often be greater than that recognized by current techniques.

Prevention of SEH/IVH and PVL in the Preterm Infant

The pathogenetic mechanisms believed to underly the development of SEH/IVH and PVL in the preterm infant brain may come into play before birth. Conditions during labour and mode of delivery may exert either a direct or indirect influence on development of SEH/IVH. Birth asphyxia or the shock associated with a mild degree of birth trauma may have a direct effect on cerebral circulation or the infant's ability to control CBF. If the mode or timing of delivery results in the infant developing RDS, then the blood gas changes of this condition may predispose to development of SEH/IVH. Thus, prevention at an obstetric level demands delivery at a time and place and by an appropriate method to minimize the risks of acute asphyxia or trauma and to ensure that the appropriate paediatric skills are available to resuscitate the infant efficiently.

In the neonatal period the principles of prevention of both anoxic-ischemic damage and SEH/IVH are similar. They must be based on avoidance of violent swings in blood pressure and consequently CBF, prevention of apnoeic attacks and maintenance of blood gas tensions within as normal a range as is possible.

The most important time during which prevention of SEH/IVH has to be acheived is the first three days after birth. The possibility of preventing SEH/IVH by administration of phenobarbitone has been investigated by Donn et al.(13) and controlled trials are currently under way at a number of centres. It is important to remember that the object of such efforts is to lower the incidence of long term handicap. Any preventive treatment which lowers the incidence of a lesion recognized on ultrasound or CT but does not influence the long term outlook is unlikely to be of any practical value.

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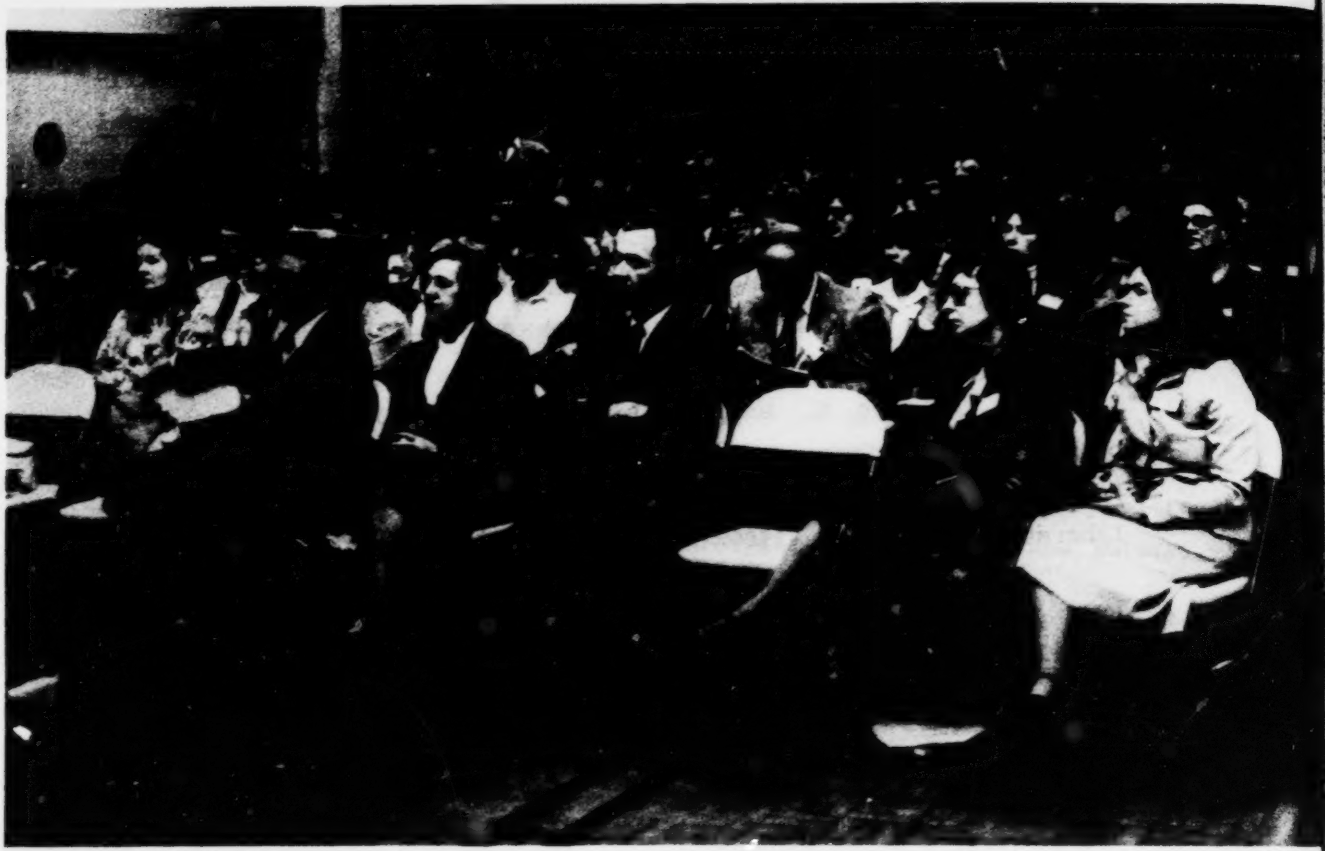
Captive Audience



FACTORS AFFECTING OUTCOME IN HYPOXIC-ISCHEMIC ENCEPHALOPATHY IN TERM INFANTS

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ABSTRACT

Forty-nine term infants were prospectively identified as having hypoxic-ischemic encephalopathy (HIE). All infants survived the neonatal period and were followed for a minimum of one year. Factors which significantly correlated with outcome included the Sarnat encephalopathy stage, and the occurrence of intractable seizures not controlled by phenobarbital alone. There was no association between the one or five minute Apgar, the need for early ventilation, the electroencephalogram, the occurrence of seizures, and the subsequent outcome. There was no significant difference in outcome for those infants who received Dexamethasone (n = 29) versus those who did not, (n = 20). Although transported infants had a greater incidence of handicap, this difference was not significant. However, a review of 97 term infants, with HIE from the regional perinatal program over a one-year period (1979) did show a significant increase in morbidity and mortality for transported infants. Further studies are required to define optimal therapy for the term infant with HIE.

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INTRODUCTION

The term infant with documented perinatal asphyxia and evidence of hypoxic-ischemic encephalopathy (HIE) has a greater chance of exhibiting neurodevelopmental sequelae than the preterm infant who is less than 1500 grams at birth.(1-6) We have previously retrospectively reviewed 95 term infants with evidence of HIE and demonstrated that 35% of these infants were either dead or had moderate to severe handicap.(5) In that study a low five minute Apgar score, the need for vigorous resuscitation, a suppression pattern on electroencephalogram (EEG), seizures occurring within the first 24 hours, and encephalopathy score as defined by Sarnat and Sarnat(7) were among the significant predictors of outcome. In addition, there appeared to be an improved outcome in the latter two years of this study. These observations, coupled with the high incidence of significant sequelae in the term infant with HIE, prompted the present descriptive study performed by prospectively identifying 49 term infants with HIE and reviewing their neonatal course and neurodevelopmental outcome to at least one year of age.

METHODS AND MATERIALS

From September 1, 1978, to December 31, 1979, 49 infants of 37 weeks gestation or greater with HIE, admitted to the Royal Alexandra Hospital Newborn Intensive Care Unit (NICU), were prospectively identified and studied. Ten (20.4%) of the 49 infants were transported.

Hypoxic-ischemic encephalopathy was diagnosed when the infant presented with an abnormal neurological examination and a history of perinatal distress as evidenced by one or more of the following criteria:

1) the documentation of intrapartum fetal distress with the recognition of abnormal fetal heart rate patterns (n = 13), with or without the passage of meconium;
2) the presence of immediate neonatal distress as evidenced by a low (five or less), one minute Apgar (n = 32), or five minute Apgar score (n = 18);

3) the need for immediate neonatal resuscitation including bag and mask ventilation (n = 16), intubation (n = 4), or the need for prolonged mechanical ventilation of greater than five minutes (n = 12).

The abnormal neurologic examination included the presence of two or more of the following:

1) alterations of the level of consciousness assessed following vigorous stimulation to rouse the infant,
2) alterations of muscle tone categorized as global hypotonia or hypertonia as well as localized tone disturbances,
3) abnormal primitive reflexes including the Moro, grasp, and suck response.

This examination was performed within one hour of admission and repeated daily. The infant was scored using the Sarnat classification(7) without EEG. Briefly, this classification involves three stages with Stage I characterized by hyperalertness, hyper-reflexia, dilated pupils, tachycardia, and the absence of seizures. Stage II includes the presence of lethargy, hyper-reflexia, miosis, bradycardia, seizures, hypotonia, and a weak suck and Moro response. Stage III is characterized by stupor, flaccidity, small to mid-position pupils which react poorly to light, decreased stretch reflexes, hypothermia, and absent Moro and suck responses.

The infants were carefully observed for seizures including either single or repetitive tonic or clonic movements as well as subtle seizures as previously described by Volpe.(8) An attempt was made to obtain an EEG shortly after admission as well as a repeat EEG prior to discharge. These EEG's were analyzed by the same individual, who was unaware of the infant's clinical status at the time of the interpretation, and were reported as previously described.(5) Computerized axial tomography (CAT scan) was done whenever possible on each child within the first seven days of admission.

The clinical management of the infant was left to the discretion of the individual neonatologist-paediatrician. Within the unit there was a general consensus favouring early vigorous treatment of seizures with an initial dose of phenobarbital 10 mg/kg followed by an initial maintenance of 10 mg/kg/day to maintain the phenobarbital levels between 2 - 3 mg %. Phenobarbital levels were obtained daily for the first three days and on the seventh day. All relevant clinical and laboratory information was entered daily on a specially-constructed data form by one of the authors.

Information regarding outcome of all children was obtained through the Neonatal Follow-Up Clinic at the Glenrose School Hospital in Edmonton, to which all surviving infants were referred for long-term follow-up. The infants were scheduled to be seen at the following ages: six months, twelve months, twenty-seven months, three and one half years, five and one half years, and eight years by a paediatric neurologist, a nurse, an occupational therapist, a physiotherapist, an infant psychologist, an audiologist, a speech pathologist, a social worker, and a consultant ophthalmologist. At each assessment a complete general physical and neurodevelopmental examination was performed.

The neurological examination included documentation of muscle tone, spontaneous movement, postural and equilibrium reactions, and reflexes. The norms for developmental levels were adapted from Gesell,(9) Illingworth,(10) and Mecham.(11) Cognitive evaluations were performed using the Uzgiris-Hunt scales of infant psychological development(12) during the first two years. At six and twenty-seven months of age, complete audiological examinations were performed.

All demographic, obstetric and neonatal data were collected retrospectively through detailed review of the hospital medical record, utilizing a medical data form constructed specifically for this purpose.*

Five categories of outcome were defined for the study population based on their most recent assessment:

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* Available from the authors upon request.

1) Normal - within the average range of development for age and no evidence of handicap,
2) Mild Handicap - variations from normal on neurologic or developmental examination without a specific diagnosis,
3) Moderate Handicap - trainable retardation, severe behavioural disorders, convulsive disorders excluding febrile convulsions, mild or moderate neuro-sensory deafness, spastic diplegia, hemiplegia or visual impairment,
4) Severe Handicap - spastic quadriplegia, severe psychomotor,
5) Death - death during hospitalization or during the follow-up period.

Only moderate handicap or greater was considered as a significant handicap, and the current report summarizes follow-up information to at least one year of age for all children.

Statistical analysis consisted of the use of Chi-square evaluation for all obstetrical and neonatal variables in relation to the asphyxia staging and outcome, with p value of less than 0.05 considered significant.

RESULTS

Patient Population - Obstetrical

The mean maternal age was 25.6 years (18 - 41) and 22 (45%) were primigravida mothers. The most frequent maternal complications included antepartum haemorrhage (12%) and toxemia (10%). There were 4 (8%) breech presentations, 2 (4%) compound presentations and 11 (22.4%) Caesarean deliveries, six of which were performed for fetal distress. Continuous internal or external fetal monitoring was obtained in 16 of these women; 13 recordings were abnormal.

Patient Population - Neonatal

There were 26 males and 23 females. All were 37 weeks gestation or greater, (mean 39.6 range, 37 - 44) by maternal dates and Dubowitz assessment.(13) The mean birth weight was 3169 grams, (range 2280 - 4450), and six were considered small for gestational age, (weight less than 10th percentile), with evidence of intrauterine growth retardation. The mean one minute Apgar score was 4.1, and five minute Apgar score was 6.6. Mechanical ventilation for either meconium aspiration (n = 1), or asphyxia which resulted in depression, with or without interactable seizures (n = 17), was required for 37% of the patients. A further 11 (22.4%) required supplemental oxygen. Seizures occurred in 34 infants (69.4%), with 25 (51%) infants developing seizures in the first 24 hours. In one infant the presence or absence of clinical seizure could not be confirmed because he had received pancuronium at five hours of age, and was maintained on continuous muscle relaxation throughout the first 96 hours, in addition to large doses of phenobarbital.

Electroencephalograms were obtained in the first week for all but three transported infants, the results of which are shown in Table 1. Of these 46 EEGs, 30 (61.2%) demonstrated a suppressed background. CAT scans were obtained in 43 infants, of which 23 were normal, five showed evidence of cerebral edema, and 15 showed evidence of an intracranial haemorrhage, including with an intraventricular haemorrhage in four and a subarachnoid haemorrhage in ten. In addition, 14 CAT scans showed evidence of periventricular low densities surrounding the anterior horn of the lateral ventricles, with two infants having hydrocephalus and two showing evidence of cerebral atrophy.

The principal forms of therapy, aside from supportive intensive care for these infants, usually involved the administration of anticonvulsants, most frequently phenobarbital. Six infants (12.2%) did not receive phenobarbital; (one of these was transported to the NICU after 96 hours of age); and none exhibited clinical seizures. There were eight infants who received phenobarbital who did not have documented seizures. They were felt to have clinically significant asphyxia, and the attending neonatologist administered phenobarbital for either "cerebral protection"(14) or to reduce the likelihood of subsequent uncontrolled seizures.(15) Six infants received pentobarbital in addition to phenobarbital, because of the presence of continuing seizures unresponsive to phenobarbital. Pentobarbital was used in a dose of 2 - 4 mg/kg q1h intravenously as required for seizures. Dexamethasone was given to 29 infants (59%), because of either suspect increased intracranial pressure or as non-specific therapy for asphyxia, with a mean initial dose of 1.0 mg/kg (0.75 mg/kg - 2 mg/kg), at a mean age of 5.8 hours (range 1 - 23). Three patients received diazepam, eight received diphenylhydantoin, and two received paraldehyde, all for persistence of seizures despite adequate phenobarbital levels.

Phenobarbital levels were monitored on days one through three and on day seven. The mean levels were 2.02 mg% at 24 - 48 hours, 2.41 mg% at 48 - 72 hours, 2.40 mg% at 72 - 96 hours, and 2.93% on day seven. Pentobarbital levels were not measured during the study.

Clinical Sarnat staging for these infants revealed that seven infants (14.3%) were Stage I, 41 (83.7%) were Stage II, and there was a single infant who was Stage III. The stage chosen was the highest observed during the first week of life.

Renal function was assessed by measuring hourly urine output and daily serum creatinine. Forty percent of the infants had oliguria as defined by a urine output of less than 1 ml/kg/hr on day one; 26% had oliguria on day two; 17% had oliguria on day three; and 4% demonstrated oliguria on day four. Ten infants did not have an elevation of creatinine above 1 mg% in the first week of life, 25 (61%) had creatinines between 1 mg% - 2 mg%, and six (14.6%) had creatinines greater than 2 mg% in the first week of life. These alterations in renal function were transient and no infants had any evidence of renal compromise at discharge.

There were no deaths in this cohort of patients and outcome data was available on all 49 infants.

FOLLOW-UP STATUS

The overall results revealed that there were no severely handicapped children. Eight (16.3%) were moderately handicapped; 13 (26.5%) had mild handicap; and the remaining children were normal. No ante-partum or intrapartum variables tested, alone or in combination, were found to be significantly related to the infant's asphyxia stage or to outcome. There was a significant association between the infant's Sarnat stage and outcome, as shown in Table 2, with the single infant who was Stage III being moderately handicapped, 7 (17.1%) infants who were Stage II showing moderate handicap, and 13 (31.7%) having evidence of mild handicap. There were five infants who were Sarnat Stage II with no evidence of seizures, and one of these infants was moderately handicapped. Thus, the outcome for infants with Stage II encephalopathy was independent of the presence of seizures. Transported infants had a greater incidence of handicap than inborn children (p = 0.059, NS), with 6 of 10 (60%) transported infants showing mild or moderate handicap versus 15 of 39 (38.4%) of the inborn infants. Thirty percent of the transported infants had a moderate handicap versus 13% of the inborn children (Chi square with Yates correction = 2.14, p = .34, NS).

Seven of 15 infants having intractable seizures not controlled by phenobarbital and requiring additional anticonvulsants were moderately handicapped, compared with only one moderately handicapped infant of 19 with single or easily controlled seizures (X^2 with Yates correction = 5.85, p = 0.0156).

CAT scan diagnosis was compared with outcome, and there was no association between any CAT scan diagnosis and outcome. The two infants with cerebral atrophy and one of two infants with hydrocephalus were moderately handicapped. There was a significant association between a low urine output on the fourth day of life and the presence of a significant handicap, with the only two infants having a urine output of less than 1 ml/kg on the fourth day exhibiting moderate handicap.

There was no significant association between any of the following variables and outcome: one or five-minute Apgar score, sex of infant, presence of seizures, time of occurrence of seizures, need for vigorous resuscitation, prolonged ventilation, initial EEG interpretation, or the presence or absence of a suppression pattern on the EEG. There was also no significant difference in outcome between the infants who received phenobarbital and those who did not, nor was there any association between the mean phenobarbital level and outcome. Although 29 infants received dexamethasone, their outcome was not significantly different from the remaining 20 infants.

The three infants who received pancuronium did not have a significantly different outcome than those who did not.

DISCUSSION

The present study has demonstrated an improved outcome for term infants with HIE compared to our previous cohort, in keeping with our previous observations of improved outcome in infants delivered more recently. This may reflect a greater awareness of this disorder and a more vigorous approach in the support of these infants. In addition to a decreased overall incidence of developmental sequelae, there was no child who died of his/her asphyxia and no child with a severe handicap on follow-up. The decreased incidence of significantly handicapped children may account for the lack of association between a number of factors which were reported to be significant in our previous study.(5) However, the Sarnat staging criteria, which were the most significant association in the previous study, still remain highly predictive for the term infant with HIE.

In the previous study, we were unable to obtain CAT scans on the majority of infants, and in this study no significant association could be found between CAT scan diagnosis and outcome. Flodmark, et al.,(16) have reported on CAT scans and prognosis for transported term infants with HIE and found that decreased brain tissue attenuation correlated with later neurologic sequelae. The same group more recently reported that CT scans were highly predictive of outcome in 62 transported term infants with asphyxia, with 11 of 15 infants having parenchymal or intraventricular haemorrhages and 18 of 20 infants with extensive areas of hypodensity having major neurodevelopmental handicaps.(6)

Only four infants in the current study had evidence of small intraventricular haemorrhages, and only one had a parenchymal haemorrhage. In addition, the hypodensity seen in the current study was not as extensive as that reported by Fitzhardinge, et al.(6)

In keeping with the results reported by Martin, et al.,(17) dexamethasone did not appear to be of any significant value in this cohort, although its use was not randomly assigned. In the present study, 87.8% of the infants received phenobarbital compared to 62% of infants in our previous cohort of term infants with HIE. Pentobarbital has been found to be a useful adjunct for the treatment of intractable seizures unresponsive to phenobarbital, perhaps because of direct depression of neuronal excitability by gamma amino butyric acid mimetic action.(18) It has been our impression that penobarbital in doses of 2-4 mg/kg/hr prn is very useful for terminating status epilepticus. Repetitive doses of pentobarbital in children may lead to hypotension as a result of a lowered systemic vascular resistance with a normal cardiac index, and requires close cardiovascular monitoring.(19)

Barbiturates may ameliorate hypoxic-ischemic brain injury by a number of mechanisms recently reviewed by Safar and Nemoto(14) and Volpe.(15) Barbiturates can reduce the cerebral metabolic rate, improve re-perfusion in relation to oxygen requirements, reduce intracranial hypertension, scavenge free radicals, suppress catecholamine induced brain hypermetabolism post-ischemia, reduce intra and extracellular edema, and have been shown to reduce neurologic sequelae in animals following pretreatment or treatment within one hour of an hypoxic-ischemic insult. In one study, however, phenobarbital in a dose of 20 mg/kg over an 18-hour period prior to an asphyxial episode did not produce any beneficial effect in a newborn monkey.(20) In spite of the fact that there are no controlled observations attesting to a protective role for barbiturates in humans, Volpe has recently recommended the early administration of barbiturates to the term infant with significant HIE prior to the onset of seizures.(15) The current report cannot shed light on this issue and a controlled trial of phenobarbital in term infants with HIE will be required to determine the benefit of such therapy.

Although others have suggested that the presence of seizures was related to poor outcome,(21) our previous cohort did not show this association, but rather demonstrated that the earlier the onset of seizures, the more significant the sequelae.(5) In the present cohort, seizures occurred in 71% of the infants compared to 68% in the previously described cohort. However, there was no significant association between the presence or time of onset of these seizures and outcome. Infants in the present study with repeated tonic-clonic seizures requiring additional anticonvulsants for seizure control had a significantly poorer outcome. Mulligan, et al.,(22) reported that infants with seizures lasting greater than 48 hours had a greater incidence of handicap, and Painter, et al.,(23) documented that infants whose seizures were difficult to control had a higher mortality. In addition, Scott, in a review of severe birth asphyxia, reported a greater incidence of cerebral palsy in children with multiple seizures,(24) and Volpe states that infants with poorly controlled seizures have more frequent and severe neurologic sequelae.(15) In view of these findings and the results of the present study, we re-analyzed the results of our previous cohort of term infants with HIE,(5) and found that infants with frequent tonic-clonic seizures unresponsive to phenobarbital and requiring additional anticonvulsant therapy had a greater frequency of significant handicap or death (21 of 41 vs. 3 of 16, $X^2 = 4.97$, $p = 0.0261$). Thus, frequent tonic-clonic seizures requiring additional anticonvulsants are a poor prognostic sign for the term infant with HIE.

In a previous study,(25) we demonstrated that 60% of term infants with HIE had evidence of renal compromise as evidenced by an elevated serum creatinine and/or the presence of oliguria. The present study confirms these observations.

Transported infants in the present study had a poorer outcome, although the numbers were small and did not achieve significance. During 1979, there was a total of 97 term neonates with HIE admitted to one of the two neonatal intensive care units in Edmonton. Eighty-two infants survived and were enrolled in follow-up, and have been seen to age one year. Table 3 reviews the outcome of these infants using the same diagnostic categories, and indicates that overall, in our area, transported infants had almost five times the incidence of significant morbidity of death as inborn babies ($p < 0.001$).

We believe that an early neurologic assessment looking for evidence of altered tone and seizures coupled with fluid restrictions, early recognition and therapy of seizures, vigorous use of mechanical ventilation for infants with evidence of respiratory compromise secondary to seizures, and good general supportive care will lead to improved outcome for term infants with HIE. Dexamethasone does not appear to be of value in the therapy of these infants. Controlled trials examining the early administration of large doses of barbiturates to such infants are urgently required to determine the role of this controversial therapy.

INITIAL ELECTROENCEPHALOGRAM IN INFANTS WITH HIE

N = 46

	NUMBER	(%)
Normal	11	23.9
Low Voltage Background	1	2.0
Low Voltage Background with Sharp or Spike Waves	3	6.5
Burst Suppression	26	56.5
Normal Background with Sharp Waves	4	8.6
Normal Background with Epileptic Focus	1	2.0

Table 1

SARNAT STAGE

TERM HIE

(1979 - 1980 - Royal Alexandra Hospital)

N = 49

	STAGE I	STAGE II	STAGE III
Normal	7	21	0
Mild Handicap	0	13	0
Moderate Handicap	0	7	1

X^2 - 11.055, p = 0.0260

Table 2

HYPOXIC-ISCHEMIC ENCEPHALOPATHY

IN TERM INFANTS: (37 WEEKS OR GREATER)
BORN IN 1979 (TOTAL ADMITTED - 97)

DEATHS AND HANDICAPS

	Total Followed	Deaths	Severe Handicaps	Moderate Handicaps	% Handicaps & Deaths
Inborn Infants	36	1	0	3	11.1%
Transported Infants	46	8	5	10	50.0%
TOTAL	82	9	5	13	32.9%

X^2 - 13.83, $p < 0.001$

Table 3

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DEVELOPMENT OF HIGH RISK CHILDREN IN THE FIRST YEAR OF LIFE

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A child in the first year of life may be at risk for many problems - developmental abnormalities, familial diseases, injuries, abuse and neglect. In today's discussion, we will concentrate on children who are at risk for developmental problems, and, in particular, children in the first year of life who are at risk of developmental problems because of adverse perinatal factors. Developmental problems rarely occur in an isolated area, so development in all areas will be considered.

Adverse perinatal factors, including preterm delivery and birth asphyxia, contribute to only one of the important groups of developmental influences. Others, which may overwhelm the effects of either a particularly good or a particularly bad perinatal course, include poverty - the greatest handicapping condition - parental, sib and peer group relationships, genetic factors, racial status, government policies regarding economic and health care matters.

An idea of the frequency with which adverse perinatal problems are associated with later handicaps, in a community, can be gained by looking at cohort studies, such as the Newcastle Child Development Survey,(1) which initially involved over 13,000 infants born in a specified geographic area during 1962 and 1963. At five years of age, information was available on 9,626 of the children initially enrolled in the study. Seventy-six (0.8%) of the children had major handicaps including Cerebral Palsy, deafness, blindness, or mental subnormality. Of these handicaps, 46.1% were due to unknown causes, 22.4% were associated with prenatal causes or familial diseases, 22.3% were associated with postnatal factors, 17.1% with certain biologic factors and 10.5% were associated with adverse perinatal factors. Obviously, in this cohort, the contribution of adverse perinatal factors to the total number of handicapped children in that area was not great. Similarly, when one looks at large numbers of children and attempts to determine the influence of single adverse perinatal factors(2) in children of mature birth weight, such as mode of presentation, delivery method and the use of forceps, major relationships are not detected. Most studies have demonstrated some association between low birth weight and poor performance on later I.Q. tests, but many factors can alter this association.

On the other hand, although perinatal factors may not be the most frequent causes of developmental problems, there is indeed a very significant "continuum of reproductive casualty".(3) If one focuses on certain sub-groups of infants or children, then the contribution of adverse perinatal factors to outcome measures may be very great. For example, Drillien studied children enrolled in a program catering to individuals with major handicaps (severe mental retardation or severe Cerebral Palsy). It is evident from her work that, although adverse perinatal factors make little contribution to the occurrence of severe mental retardation, they do make a major contribution to cases of severe Cerebral Palsy. If one looks at the contribution different neonatal problems make to later outcomes, then very significant relationships are noted between the occurrence of intracranial haemorrhage and neonatal seizures, and later outcome, with far less relationship noted between such areas as birth weight-gestational age category, or the Apgar score.

The changes in perinatal mortality statistics during the past few decades are very striking. For example, in the province of Alberta the perinatal mortality rate fell from 19.1 stillbirths and neonatal deaths per thousand total births in 1971 to 12.0 in 1979. At the same time, the neonatal mortality rate fell from 12.7 deaths during the first 28 days of life per thousand total births in 1971 to 8.1 in 1979. Fortunately, in spite of early scepticism regarding this, similar striking changes have occurred in morbidity. The changes have been in both frequency and type of morbidity detected in follow-up.

Early follow-up studies such as that done by Lubchenco,(4) who looked at a group of 10 year olds who received "premature infant nursery care" in

1947-50, demonstrated a handicapping condition rate of over 70%. Drillien,(5) who looked at children born in 1952-53, demonstrated a similar handicap rate in survivors. More recently, several studies have demonstrated handicap rates in the 6-8% range, particularly if one looks at entire groups of low birth weight infants or infants representative of intensive care nursery populations. If one selects very small groups of ultra-high risk infants from intensive care nursery populations, then the frequency of handicaps is higher. In Calgary, where only ultra-high risk neonates are selected for follow-up from the over 700 patients admitted to the neonatal intensive care unit, Foothills Hospital, per year, handicap rates near 20% are the rule.

In the past, frequent handicaps noted in survivors were Cerebral Palsy (Spastic Diplegia) and blindness. The changes in the epidemiology of Spastic Diplegia have been described in several communities, and it is evident that Spastic Diplegia occurs in smaller proportions of intensive care nursery survivors than was the case a short time ago. Retrolental Fibroplasia was an overwhelmingly common cause of blindness in children cared for in intensive care units in the early 1950's, but, more recently, it has become a less frequent cause of blindness, though it remains significant indeed. More common problems in intensive care nursery survivors today are major pulmonary problems, and frequently more subtle neurological problems and behaviour disorders.

In order to consider the development of infants who have had adverse perinatal factors, one can begin by selecting from the relatively large number of patients admitted, for example, to an intensive care nursery, those who are at greatest risk of later abnormalities. During the period January 1, 1977, to April 30, 1981, 2,354 infants were admitted to Foothills Hospital Intensive Care Nursery. During most of that period, it was decided that children judged to be at very high risk would be those that weighed less than 1.5 kilograms at birth, those who required assisted ventilation in the neonatal period, those with congenital infections such as Rubella, those with severe birth asphyxia and those with neurologic abnormalities noted in the neonatal period. Eight hundred and fifty-six of the 2,354 children admitted fit one or more of these criteria, and 624 of the children survived. Because our resources for follow-up of these children have been very limited, it has been possible to assess only 300 of these survivors to date. Initial data analysis regarding the antecedents of developmental and other disabilities in this group have allowed us to use more restrictive criteria, so that more recently the children regularly enrolled in the follow-up program include those who weighed less than 1250 grams at birth, those with complicated ventilator courses, those with congenital infections, those with neurological abnormalities and finally those who have Retrolental Fibroplasia.

Enrolling such groups of children in a follow-up program is a straightforward and manageable approach, but it may be possible, on the basis of early evaluations, to be even more selective. During the past five years, a physiotherapist in the follow-up program and an ophthamologist have examined all of the children who met the follow-up program criteria, prior to their discharge from the intensive care nursery. The results of the assessment by these two individuals are used to assess priorities for assessment within the children who fit the criteria. This utilization of established criteria, plus priority assignment on the basis of functional assessment, would appear to be more efficient than using either of these techniques alone.

In order to evaluate follow-up studies and follow-up results, one must be familiar with "ICN experience". In the not-too-distant past, there was frequently a "hands off" approach to the care of the ill preterm infants. Now, the environment has become one described by Brazelton(6) and others as "relentlessly intrusive". There are rational reasons behind most of the intrusions, many of them related to the performance of monitoring events, measuring blood gases, hematocrits, ultrasound examinations and other lab work. Nevertheless, there is a growing body of evidence to suggest that not only are there associations with certain perinatal factors and later outcomes, but also the fact of spending a long time as a patient in an intensive care nursery may have some of its own adverse effects.

The initiation of follow-up assessments in the intensive care nursery by individuals familiar with the intensive care nursery experience provides optimal diagnostic ability, and allows certain sensitivity leading to the possible early introduction of changes beneficial to the infants. Attention to the linkage and ongoing communication of neonatal intensive care nursery and follow-up program personnel, in my opinion, is an integral part of modern perinatal care.

Many stimulation programs or very early intervention programs have been introduced into intensive care nurseries, usually emphasizing motor, auditory, visual or tactile stimulation. One such program(7) includes having individuals spend periods of time providing pleasurable touch for infants on a frequent basis, for example, hourly. Another example would be the use of waterbeds.(8) In many nurseries staff members provide pleasurable non-nutritive sucking experiences(9) for the infant, and in others a great deal of attention is paid to infant feedings, laboratory tests, etc., on a "demand basis", the limits being set by the infant. The most successful intervention programs include capitalizing on each infant's individual abilities and states, and promoting interaction between the parent and infant.

In the Alberta Children's Hospital Perinatal Follow-up Program, in addition to the early physiotherapy and opthalmologic assessments mentioned, some children receive a Brazelton Neonatal Behavioral Assessment.(10) This test was initially developed for use in healthy, term infants, but Brazelton and his colleagues have modified this tool for use in preterm infants. The assessment is used to assess the behavioural state, but, perhaps more importantly, it can be used as a form of effective communication between parent and health professionals, it can be used as an intervention involving demonstrating to parents abilities of the infant that they might have not otherwise recognized, and perhaps it allows a demonstration to parents of the fact that at times an infant will be receptive to stimulating activities, and at other times, he or she will become only frustrated and angry if stimulating activities are pushed.

Follow-up assessments in the Perinatal Follow-up Program at the Alberta Children's Hospital begin at four months adjusted age. Children may be seen before that time if the parents specifically request it or if there are special concerns regarding ophthalmologic problems, which must be followed very regularly early in life since there are critical times regarding the development of Retinopathy of Prematurity and the institution of appropriate therapy for advancing retinopathy.

When the infants are seen at four months adjusted age a variety of types of assessment are performed. These include, first of all, a health history and nursing assessment, which includes determination of various areas of parental concerns and infant development, immunization status, the use of medications and various health care and education resource utilization. Many parents whose infants have neurologic problems may initially present behavioural complaints, such as "the child is always jittery", or "always fussy", that the child "is too stiff to handle", or that the infant "stands too well". There are differences in development of infants who were very low birth weight, and in those born at term, including EEG differences, differences in visual attentiveness, differences in perceptual development, and probably differences in other areas as well. Information is only now being collected in these areas. In addition to a nursing assessment at four months adjusted age, each child will also be seen by a paediatrician for physical examination, by an ophthalmologist and orthoptist, a physiotherapist and an audiologist.

Several findings have been reported regularly on physical examination in children seen in follow-up. These include, in the earliest assessments, descriptions of the typical long, narrow head shape and somewhat unusual facial features including prominent eyes and cheeks, that are common to those working with low birth weight infants. The facial features usually lessen markedly by four months adjusted age.

In the recent past, many children in the intensive care nursery were intubated for long periods of time by the nasal route. This has been associated with asymmetry of nostril size which persists over time. Now, in our program, children are intubated by the oral route, and no nasal abnormalities are detected. One now sees relatively high, sometimes grooved palates, and frequently the upper teeth, particularly the upper central incisors, are somewhat mottled and hypoplastic. This may be related to oral tracheal tube positioning, with pressure on the upper gum margin during the neonatal period.

Skin defects and scars are perhaps not the most serious types of problems found on physical examination, but some scars can be quite deforming, requiring surgical revision, and many times major scars may be somewhat avoidable. It is of interest to note that, in our program, very significant scars have been seen secondary to chest tube placement with purse-string sutures utilized. Some scars could interfere with later breast development if thoughtlessly placed.

Other abnormalities frequently noted on physical examination include pulmonary abnormalities, including abnormalities of chest shape, presence of respiratory difficulties and abnormal breath sounds, and, less often, findings of Patent Ductus Arteriosus. Most of the children have umbilical hernias, or at least a flat umbilicus and some have very irregular heel shapes, occasionally with cystic lesions, secondary to early heel sticks. 63.6% of the children are normal on physical examination at twelve months of age, and by four years of age 80% are normal.

The health problems are, for the most part, pulmonary. They include recurrent lower respiratory tract infections, including bronchitis, broncholitis, and pneumonia and many have recurrent episodes diagnosed as "croup" or "asthma". G.I. illnesses, the viral exanthema of childhood and trauma do not appear to be excessively frequent in the group of patients we are following. There is some ongoing mortality, usually related to respiratory or neurologic problems or SIDS.

In the Alberta Children's Hospital Perinatal Follow-up Program, a great deal of attention is paid to the assessment of muscle tone, the disappearance of primitive reflexes and appearances of postural reflexes and motor development. In this regard, a physiotherapist performs the test of Gross, Motor and Reflex Development, which was also performed first in the intensive care nursery before the child was discharged, and which is performed serially during the first few years of life. On the same occasions, the Amiel-Tison Longitudinal Neurologic Evaluation is performed by the paediatrician. This assessment tool is designed as a longitudinal evaluation to be done by a non-neurologist, and it is by no means to replace a standard, complete neurologic examination. On the basis of these two assessments, children can be divided during the first year of life into three groups - those who are normal throughout, those who are abnormal throughout, and a particularly fascinating group, those who are initially abnormal but later become normal by 12 months of age. It is in the latter group of children that subtle behavioural and learning problems may surface at school age. Capute(11) and others are in the process of evaluating the use of the primitive reflex profiles alone in predicting later outcomes. Early in the course of child development, the paediatrician in this program also performs the Gessell Development Screening Inventory,(12) which allows assessment of development in several areas including growth, motor, fine motor, language, adaptive and personal social areas.

Major hearing problems are not as common in populations of intensive care nursery survivors as they were some years ago. There are, however, many perinatal events which expose a child to risk for hearing difficulties. These include ototoxic drugs such as Kanamycin, Gentamycin, Lasix, and Ethacrinic Acid. In addition, hearing deficits may occur in association with borderline bilirubin levels, or mild Kernicterus with hypoxic episodes, with certain congenital defects, and the same types of racial and societal factors that are associated with a high frequency of prematurity.

The noise levels in intensive care nurseries are high. In the incubators used at Foothills Hospital, the constant noise level is 60-70 decibels and it is slightly higher if oxygen, CPAP or phototherapy are running. Noise-induced hearing loss may occur in industry in individuals exposed to noise levels above 80 decibels. It is not clear how important this noise is to preterm infants, though we have noted occasional children who have "high frequency dips", an abnormal audiogram pattern that is associated with only noise-induced injury.

In the Alberta Children's Hospital Perinatal Follow-up Program, six of the 300 children seen to date have hearing aids. Three of these children did not have major problems. In all six, hearing aids were provided at an early age, and satisfactory progress has been noted in the speech and intellectual development of the three. The other three had multiple handicaps, including visual handicaps in two cases, and neurological handicaps in three cases.

The progress in these children has not been as good. Mild conductive hearing loss associated with tympanic membrane mobility abnormalities and "serious otitis" is very common in the preschoolers seen in this program.

Speech abnormalities also are quite frequent, more so in some follow-up programs than in others. In this program, 79% of the children have normal language development at 12 months of age, 6.2% are abnormal, and in 14.8% speech abilities are "suspect". The variability from program to program may relate to different speech-testing techniques. In this program, standard speech pathology testing tools are administered by a speech pathologist, and information on language development is also derived from other sources, including a language sample that parents provide and a "parent inventory". The inventory used at 12 months of age in this program is the Minnesota Child Development Inventory. A review of our experience in this area demonstrates high correlation between expressive language scores on the parent inventory and those noted by the speech pathologist. The speech pathologist's assessment, of course, provides much parental education and testing not only of expressive language development but also of receptive language development, assessment of articulation and voice quality.

As was noted earlier, very major ophthalmologic difficulties, associated with Retrolental Fibroplasia, were very common in infants born in the early 1950's. Retrolental Fibroplasia remains a difficult problem, but there have been encouraging developments in the area. In particular, consideration of Retinopathy of Prematurity of different grades, and of the feasibility of intervening, using cryotherapy at a critical period, offers results encouraging to the preservation of useful vision in children who would otherwise progress to severe Retrolental Fibroplasia and, perhaps, major visual impairment.

In addition to Retinopathy of Prematurity or Retrolental Fibroplasia, a variety of other ocular problems may occur in infants who are "at risk" because of adverse perinatal problems. The incidence of problems varies between centres and between periods of time, because of variations in testing abilities and certain therapies. Ellingham(13) noted that in a population of high risk children in New Zealand, 5.9% had visual defects and none of these appeared associated with major neonatal problems. McCormick(14) of Vancouver noted that strabismus occurred in 14% of low birth weight infants and in only 3.6% of a group of controls.

In our program, refractive errors and strabismus appear quite common in children between one and two years of age, but only four children are blind: two blind in both eyes and two blind in one eye with some functional vision in the other eye. Of the children blind in both eyes, one is blind because of Retrolental Fibroplasia and one is blind because of optic atrophy, presumably associated with hypoxia. It is noted that the occurrence of ophthalmologic problems in children at four months of age is significantly associated with the occurrence of ophthalmologic problems in the follow-up at two years or four years of age.

The occurrence of growth problems in preterm infants requires special attention. In this program, a separate study is being carried out to determine the growth and dietary status of a large group of preterm infants in comparison to information collected from "control children" at the City of Calgary Community Health Clinics. Although the follow-up clinics' population appears at a lower range of normal when height, weight or head circumference are plotted in terms of chronologic age, the situation is different with growth nearer the 50th percentile, if the measurements are considered according to adjusted age. By two years of age most children have had significant catch-up growth, though this is not as frequent in those who were small for gestational age, in those with Bronchopulmonary Displasia or in the children with neurologic problems. Feeding difficulties, such as colic, do not appear to be particularly common in the high risk infants seen in this program.

Children noted in follow-up to have problems requiring intervention are referred for further investigations, including social work, psychology and occupational therapy (which have recently become a regular part of the follow-up program) and for neurological assessments as well. In addition, very early referrals to treatment programs are made where indicated. The practicality and desirability of early referrals is clear, but the objective results have not yet been confirmed. Many questions remain regarding the association between early developmental measures and later outcomes measures. For example, it has been said that there is little correlation between early testing which usually concentrates on sensory-motor activities, and later testing which usually includes more abstract areas, more direct measures of cognitive development. Nevertheless, there are some correlations. In particular, the finding of neurologic abnormalities early in life does correlate with the finding of neurologic abnormalities later in life. The early detection of neurologic problems permits the institution of some means of intervention which can at least prevent aggravation of existing handicaps by development of inappropriate behavioral patterns, motor activity, or preferred positions.

The information collected in the Alberta Children's Hospital Follow-up Program is regularly passed on to those responsible for providing care to high-risk mothers of infants, and follow-up activities are seen as an integral part of the perinatal care process. Failure to recognize the need for some type of long-term outcome monitoring, in association with the massive and growing expenditures of human and technologic health care resources in the area of high risk obstetrics and neonatology, would be irresponsible.

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MYOMETRIAL EFFECTS ON FETAL DEVELOPMENT

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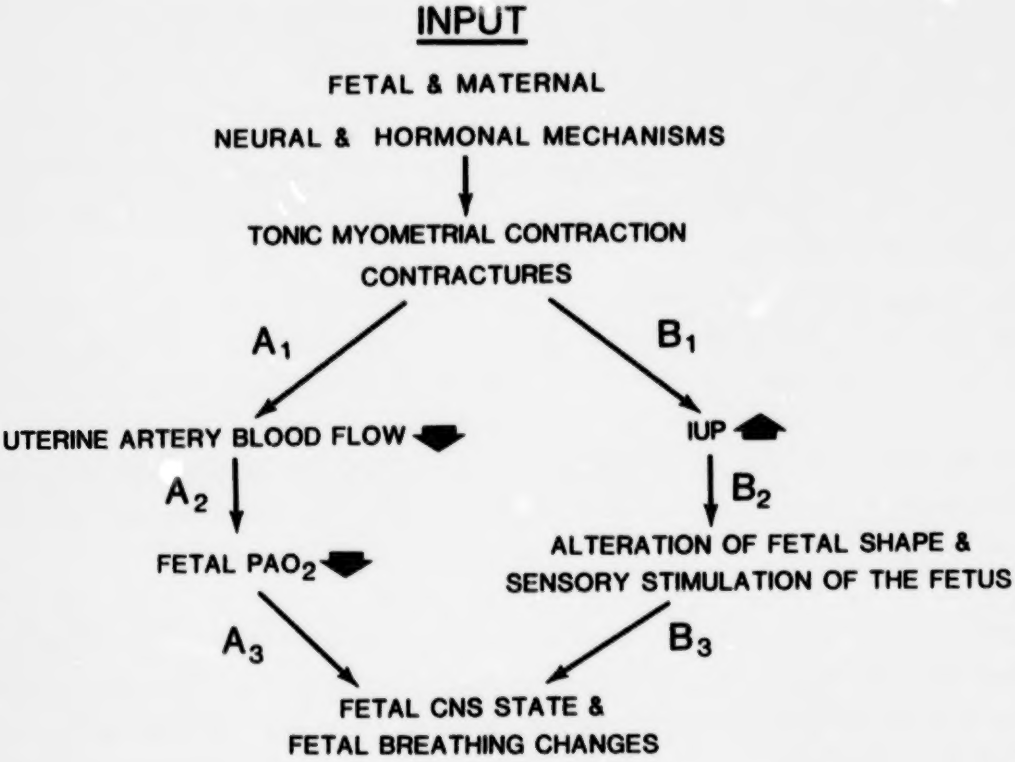
Maternal Influences on Fetal Development

As Dr. Wigglesworth has described to us in his presentation, fetal development involves an internally coded, programmed capacity interacting with environmental influences. At any one critical time for growth, external influences may modify development significantly. These may be influences that produce clearly observable physical manifestations, such as the sequelae to thalidomide, but, in addition, it is becoming progressively clear from our knowledge of neurochemistry that marked alterations may occur in neural mechanisms that do not present with externally manifested changes in phenotype. These modifications may be minor and temporary but the possibility exists that they may be more significant and permanent.

Androgen sterilization of neonatal rats by administration of androgens to the newborn female rat for a very few days after birth will permanently alter hypothalamic function in the exposed female.(1) I wish to discuss our recent work on myometrial activity throughout gestation in two experimental models - the pregnant sheep and the pregnant monkey. In the sheep, we have demonstrated that the uterine muscle is undergoing tonic, episodic contractions lasting 5-15 minutes and occurring every 30-120 minutes. In order to distinguish this form of myometrial activity from the shorter-lived expulsive efforts of labour and delivery, we have called these episodes "contractures".(2,3,4) Contractures are associated with a fall in fetal intravascular PO_2(3) as well as changes in fetal sleep state and fetal breathing.(4)

We propose the following hypothesis. Tonic myometrial activity affects fetal breathing movements and fetal sleep state by one or other of the two mechanisms demonstrated in Figure 1.


INPUT
FETAL & MATERNAL
NEURAL & HORMONAL MECHANISMS
TONIC MYOMETRIAL CONTRACTION
CONTRACTURES
A1
B1
UTERINE ARTERY BLOOD FLOW
IUP
A2
B2
FETAL PAO2
ALTERATION OF FETAL SHAPE &
SENSORY STIMULATION OF THE FETUS
A3
B3
FETAL CNS STATE &
FETAL BREATHING CHANGES

A_{1-3} and B_{1-3} represent two possible mechanisms whereby contractures may affect fetal breathing and fetal sleep state. (Previously unpublished figure, copyright P. W. Nathanielsz.)

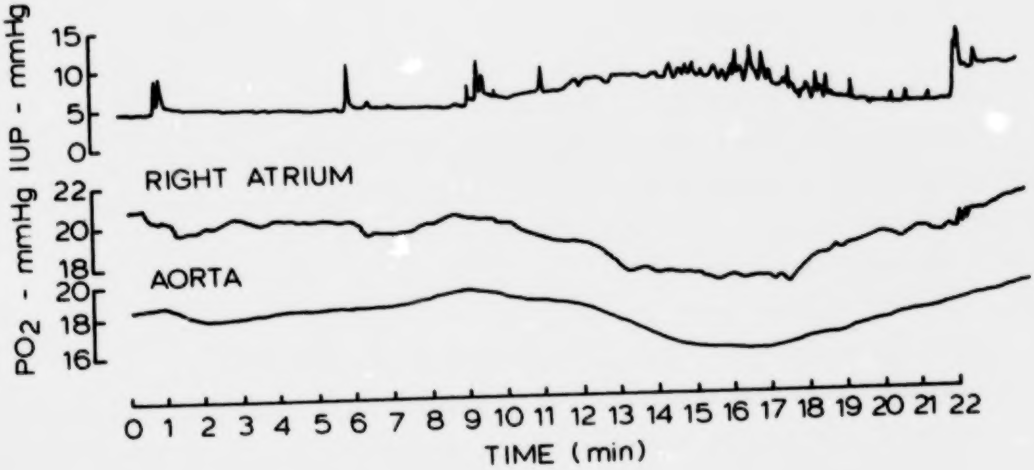
Figure 1

Studies in the Sheep

Effect of tonic myometrial activity on fetal oxygenation

Contractures cause a fall in fetal intravascular PO_2 (Figure 2). The fall may be 5 mm Hg PO_2 or occasionally even more. Such a fall represents a fall in fetal blood oxygen content of 25-35%, since in the physiological range fetal PO_2 and oxygen content are linearly related.(5) Artificial depression of fetal oxygenation to the same extent by occluding uterine blood flow for short periods of time by inflating cuffs placed around the uterine arteries will produce inhibition of fetal breathing.(6) We have demonstrated that contractures do impair uterine blood flow.(7)

These observations may have both physiological and pathophysiological consequences. Since it has been demonstrated that changes in fetal neural activity can be produced by changes of fetal oxygenation of the extent produced by contractures, we propose that the continuous variability of fetal PO_2 may be an important input to the maturing brain. From the pathophysiological viewpoint, excessive amounts of contracture activity would lead to continuously depressed fetal PO_2 with the potential for increased anaerobic glycolysis and lacticacidemia which has been suggested as a major cause of brain damage.(8)



Simultaneous measurement of IUP and right atrial and aortic arch PO_2 in a sheep fetus, gestational age 113 days, 5 days postoperatively. Fetus died during delivery following induction of labor with adrenocorticotropic hormone at 123 days' gestation. (Reproduced with permission from Jansen, et al., 3.)

Figure 2

Effect of tonic myometrial activity on fetal sensory input

Contractures are accompanied by a marked deformation of the fetal thorax(4) and probably other fetal parts. Some investigators have experienced difficulty with this concept. Illustrations of the fetus in utero show it surrounded by fluid and, since fluid is incompressible, many people refuse to accept that a contracture could deform the fetus. However, direct observation of the pregnant human, monkey or sheep uterus clearly shows that the uterus is in contact with the fetus. In the last third of gestation the amount of amniotic fluid (and allantoic fluid in addition in the sheep) is decreasing daily and does not at any time completely surround the fetus. Therefore, when the uterus contracts, the fetus is squeezed.

In the last third of gestation both the afferent and efferent limbs of the fetal nervous system are well developed. We hypothesize that the sensory stimulation of the fetus caused by contractures may have both physiological and pathophysiological effects on the fetus under different circumstances.

A normal physiological frequency and extent of contractures may be important in the patterning of fetal neural development. This idea would be in keeping with the effects that have been demonstrated in the visual pathway of newborn monkeys when the normal pattern of visual input has been removed.(9)

The pathological consequences of too much contracture activity could result from the abnormal fetal sleep-wakefulness patterns that would result. In a very thought-provoking study, Mirmiran and his colleagues have shown that, reducing by only 50% the amount of time spent by newborn rats in Random Eye Movement (REM), sleep for the short period of 7 - 21 days of neonatal life has profound effects on learning behaviour and sexual performance when the rats reach adulthood.(10) The critical periods of neural development that occur between day 7 and 21 of neonatal life in the rat occur during intrauterine life in the human. Contractures switch the fetus from REM to non-REM sleep, and therefore would tend to produce a reduction in time spent in REM-sleep, with the possibility of consequences as significant as those observed in the newborn rat. This hypothesis should be investigated.

Experimental Work in the Monkey

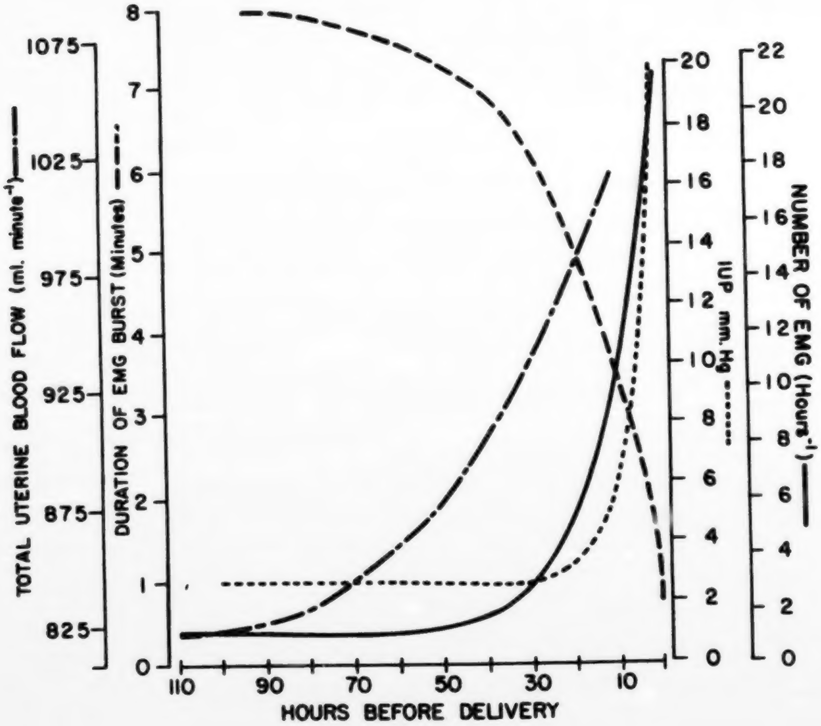
Invasive techniques to study the occurrence of contractures and their effects on the fetus are clearly unethical in the human. Many pregnant women do report 'uterine tightening' throughout a large portion of the latter part of pregnancy. These may be the so-called Braxton-Hicks contractions.

In the chorionic pregnant monkey preparation, prolonged bursts of myometrial activity do occur in the latter part of pregnancy.(11) These studies are, however, in their preliminary stages and the pregnant non-human primate uterus appears to show both similarities to and differences from the pregnant sheep myometrium.

Alteration of Tonic Contracture Type Activity to the Short-Lived Labour and Delivery Contractions in the Sheep

We have followed both the electrical activity of myometrial contraction and the pressure changes generated by these events through the last third of gestation and through both spontaneous and induced labour and delivery in the sheep. Several early reports stated the observation of intrauterine pressure (IUP) changes in the pregnant sheep at term did not demonstrate any significant pressure increase until less than 24 hours before delivery. In both the sheep and monkey, our studies show that continuous recording of myometrial electromyogram (EMG) permits clear anticipation of both normal and prematurely induced delivery.

In the sheep in both term and premature delivery (induced by the infusion of ACTH to the fetus), the mean duration of individual EMG bursts decreases (Figure 3). This may be due to the appearance of a qualitatively new type of input to the uterus that results in a different pattern of muscle stimulation or a change in response of the muscle itself (possibly by modification of its receptor population). The next change in the myometrial EMG pattern is that the individual bursts become more frequent and finally the IUP change associated with each burst increases.(6) Garfield, Sims and Daniel(12) have demonstrated in the sheep that gap-junctions between the individual myometrial cells appear during parturition and disappear after delivery.



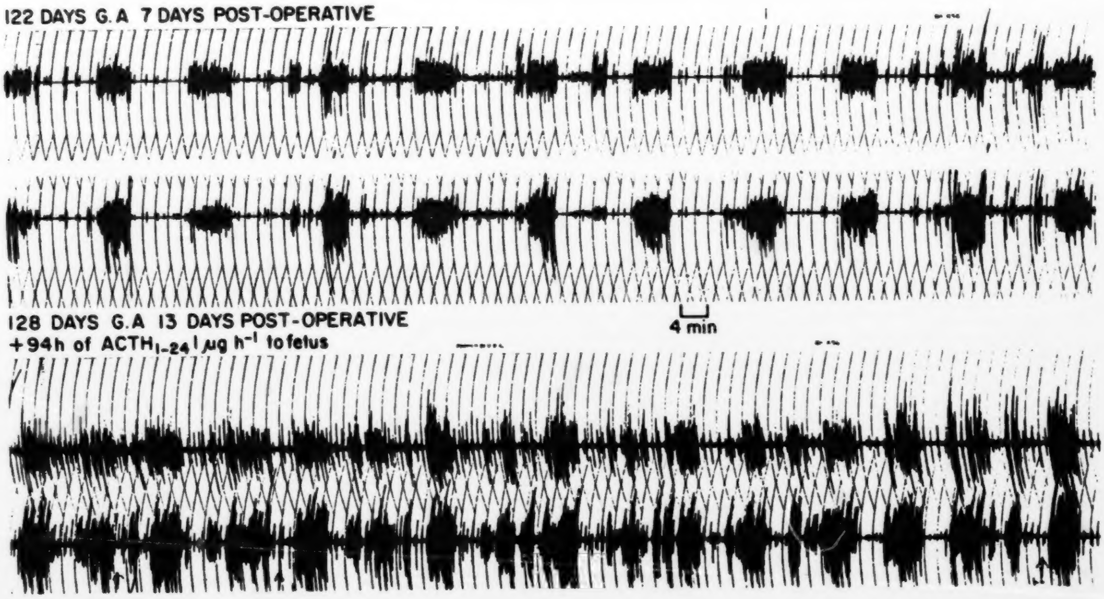
Changes in total uterine blood flow, duration of individual EMG burst (min), number of EMG bursts per hour and intrauterine pressure (IUP) increment associated with each contracture in a pregnant ewe in which premature delivery was induced by the infusion of 1 ug ACTH$_{1-24}$.h^{-1} to the fetus at 120 days gestation.

Figure 3

A significant observation in the animals studied in premature labour following the infusion of ACTH to the fetus was that total uterine blood flow increased before the changes in the myometrial activity began. The uterine blood flow was measured with electromagnetic flow probes placed on each middle uterine artery. The value taken for flow was the maximum value between episodes of myometrial activity as recorded by both EMG and IUP traces. This increase in basal uterine blood flow can be seen as a compensation mechanism that will help to offset the decrease in overall uterine perfusion that occurs during the increasing myometrial activity. It correlates well in time with the increase in maternal estrogens.

Effect of maternal posture

In the sheep, contractures are very frequently associated with a change in posture (Figure 4). It appears that the ewe can 'respond', either consciously or unconsciously, to the episode of myometrial activity, thus showing some similarity perhaps to pregnant women who are conscious of Braxton-Hicks activity.



Continuous recording of intrauterine pressure (IUP) (upper trace) and myometrial EMG (lower trace) from a pregnant ewe at 135 days gestation and 10 days post-surgery. The pressure transducer was fixed to the side of the cage and thus the sudden large upward deflection occurs when the ewe stands and the downward deflection occurs when the ewe lies down. IUP changes associated with contractures are superimposed on these postural changes.

Figure 4

We have noted that total uterine blood flow measured using electromagnetic flow probes increases when the ewe lies down. This increase more than counteracts the fall in uterine blood flow that is associated with a contracture, if one is occurring at that time. A careful analysis of the events occurring at the time the ewe lies down requires the disassociation of the effect of any contracture present from the effects of posture on uterine blood flow. When these two effects are dissociated, there is a clear increase in uterine blood flow as a result of lying down. This observation is sound evidence for the advisability of bed rest for pregnant women.

Further studies of the mechanisms that produce these gradual changes in uterine blood flow and myometrial activity will yield information of value in improving our understanding of the onset of term and premature labour as well at postmaturity. Rational therapies of these conditions can then be more usefully instituted.

Recommendations

These basic investigations of myometrial activity lead to the following proposals for future work, both experimental and clinical, that is required in these areas.

1. Control of Myometrial Activity

Experimental Information Required

Precise studies are required to define normal patterns of uterine muscle activity in primate and non-primate animal models. A better understanding is needed of the control of basal tonic myometrial activity (contractures) throughout gestation and its transformation into effective expulsive contractions of labour.

Clinical Requirements

In the development of non-invasive methods of assessing the various forms of myometrial activity in the human the myometrium should be a large enough muscle from which to produce useful electrical recordings.

Benefit

Knowledge of the various mechanisms that control myometrial activity will aid the prevention and management of premature labour, disco-ordinate uterine activity and post-maturity.

2. Fetal Neurological Maturation

Experimental Information Required

Animal studies using invasive techniques should be conducted to assess the importance of tonic contractures on normal fetal development.

Clinical Requirements

Non-invasive techniques should be used to obtain data that will improve our understanding of fetal neural maturation, as expressed by various features of fetal activity.

Benefit

The role of maternal influences on normal fetal development requires elucidation to offer important constructive suggestions for antenatal care.

3. Pathological Myometrial Activity - Causation and Consequences

Information Required

Invasive techniques should be used in the primate and non-primate animal model to assess the effects of various agents and activities such as caffeine, smoking and maternal stress on myometrial activity and fetal development.

Benefit

A better understanding of the pathological mechanisms will improve diagnoses and management.

SUMMARY

Low grade tonic myometrial activity throughout gestation has been demonstrated experimentally in several experimental animal species. This activity is probably related to Braxton-Hicks contractions. In the sheep, these uterine contractures are associated with a fall of up to 25-35% in fetal intravascular PO_2, occurring probably as a result of a decrease in uterine blood flow during the contracture. In addition, the contracting uterus produces distorting forces on the fetus. When the fetal sleep state is REM, a contracture will usually switch the fetus into non-REM sleep.

We hypothesize that these contracture-related changes have an important physiological role to play in the patterning of the input to the developing fetal central nervous system. In addition, excessive frequency or extent of contractures may produce pathological neural development. This possibility merits further testing and may, in part, constitute the mechanism whereby such agents as caffeine, smoking, alcohol and maternal stress affect fetal development.

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ASSESSMENT OF THE FETUS AT RISK

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I hope to go through with you the various methods that we have of assessing the fetus in utero, starting at the very beginning of the pregnancy and working our way towards the delivery.

Risk Scoring

We anticipate areas of potential high risk, and pay particular attention to those cases only. In the Royal Alexandra Hospital, we use a system of scoring which is colour-coded. A red flag means that there is a risk of 10x normal perinatal mortality. The golden flag is 5x normal perinatal mortality, and so on. There are a series of medical or obstetrical complications of unquantifiable risk which are colour-coded green.

Ultra high risk includes gestations of (1) less than 32 weeks, (2) suspect intrauterine growth retardation (IUGR) or placental insufficiency, (3) polyhydramnios with a single fetus, (4) any insulin-taking diabetic, (5) pregnancy at any stage where there is antepartum bleeding and (6) the multiple gestations. These are the patients who will benefit most from meticulous attention to early diagnosis and treatment. Patients with 5x normal perinatal mortality include (1) erythroblastosis, which, fortunately, in Alberta is a disease which is almost disappearing and, (2) the transition group of gestations, where the baby will be born between 32 and 35 weeks of gestation.

Intrauterine Growth Retardation

One of the things that I would like to talk about right now is intrauterine growth retardation. Antepartum, this is associated with intrauterine deaths. Intrapartum, it may be associated with asphyxia or stillbirth and there are various neonatal sequelae. First, we have to be able to make the diagnosis. We have to suspect that this pregnancy really is growth-retarded, and consider genetic potential. I think it is very important when you see any patient, to find what her own birth weight was, and what birth weights she has produced with her own previous pregnancies and what weights were her siblings. Determine whether that family produces big babies, small babies, or whatever.

Etiology

Environmental factors can be very important, and most important of all of these are behavioural, such as smoking (as Dr. Rush found), alcoholism and other drug addictions that we know of: caffeine, Talwin (which is a very common addiction in Edmonton, for instance) and other narcotics. Other factors may have an effect or an impact on uterine placental blood flow, such as vascular complications associated with diabetes and vascular complications associated with hypertensive disorders.

History

We have to know that dates are accurate. It is very important, for instance, to know when the LMP occurs. (About 25-30% of all our patients haven't a clue.) You may get further information upon finding out the date of conversion of a negative to a positive pregnancy test. Check up on menstrual cycle length. Listen to the patient. Find out how the patient thinks she is growing during the course of this pregnancy. Does she think she is small-for-dates, or does she think she is large-for-dates, or about the same size she was before in the previous pregnancy? What are her comments upon such factors as fetal movement? The Cardiff score is a maternal measure of fetal activity, i.e., the time it takes for the fetus to make 10 vigorous movements during the day, extending the count to 12 hours, if necessary. If she had 10 movements by 10:00 one morning, 10 movements by 11:00 the next morning, 10 movements by noon the next day, it is a gradual pattern of decreasing fetal activity that might be quite significant, and it is well worth while checking into. Obviously, if you have an intrauterine death, you lose all fetal movement. Mothers often will say that they have not felt movement for three or four days, but they did not think this inactivity was significant, and did not want to bother the doctor. By the time they do come in, they have a macerated fetus and there is nothing that can be done.

Physical Examination

I would like to make a plea here for another very simple and objective advance in obstetrical care of the past five or ten years - ordinary tape measure to accurately assess symphysis-fundal height. The rule that you use is that, for each completed week of gestation, there is about one centimeter's growth. Normal growth velocity is one cm/week. If the growth is less than that, you should be concerned about possible intrauterine growth retardation. When the growth is more, you have to think about possible polyhydramnios or multiple gestation. The measure is simple and reproducible from observer to observer. You can readily detect incorrect dates and all the growth disturbances, multiple gestations, at a very early stage in gestation. Early detection then permits more detailed investigation and attempts at simple treatment before the patient or the fetus reaches a critical phase.

Uses of Ultrasound

I will be mentioning several times the advances using ultrasound, and as I said, if the paper tape measure has been one of the most major advances in recent obstetrics, ultrasound - both static and dynamic ultrasound - has to be the next most major advance. It is really phenomenal the varied and yet detailed information we can now obtain. Most simple is the biparietal diameter. Serial head diameters of so many millimeters or centimeters may be plotted out according to the weeks of gestation to develop growth curves. Knowing these curves, BPDs between 14 and 26 weeks will permit you to assign or confirm a due date to patients, even in those that do not have a LMP. What is more important, you can search for evidence of intrauterine growth retardation, head-sparing and so on, by supplementing BPD with abdominal diameters.

Diagnosis

How may we make the diagnosis of intrauterine growth retardation? We can use the paper tape measure to first identify those patients who appear to be inappropriately small-for-dates. Then with early ultrasound, a single biparietal diameter between 14 and 26 weeks will either confirm our menstrual data or suggest new dates. To check, we repeat the BPD in two to four weeks' time. In this way, we confirm our first impression and establish fetal growth velocity. We can also measure total intrauterine volumes, but this is primarily a research tool, and not readily available to most clinicians. Frankly, the total intrauterine volume does not tell you anything that a simple tape measure would not, if you just measure from the symphysis to the top of the uterine fundus. Finally, up to about 37 weeks, the fetal head is usually uniformly larger than the belly. At about 37 weeks, the two are equal, and beyond 37 weeks, the belly becomes larger than the fetal head. Study of the absolute diameters and comparison of these ratios may help to differentiate a constitutionally small baby from a child with IUGR and head-sparing, if the duration of pregnancy is known.

PRE-ECLAMPSIA

There are ways now that one can predict which patients will become pre-eclamptic. We happen to use an automatic blood pressure monitor. In the rollover test, the patient's BP is taken every five minutes for an hour in different positions; i.e., every five minutes starting with thirty minutes on the left side, fifteen minutes on her back and fifteen minutes back on the left side again (i.e., a total of twelve measurements). Unfortunately, this is time-consuming. Furthermore, BP is remarkably labile, therefore our use of a mechanical device. The rollover test will actually predict, in a very high percentage of cases, those patients who will subsequently develop pre-eclampsia. For the prediction of pre-eclampsia, one should look for a rise in the diastolic pressure of at least 20 mm Hg in a patient when she turns onto her back. Now, some patients have a different response in a rollover test, because when you turn them from their side onto their back, instead of the diastolic pressure rising, their systolic pressure will actually fall, and these are the patients that have sufficient caval occlusion that they develop a true supine hypotension. Of course, we all know if we talk to patients, they do not like to be flat on their backs.

Another very simple test that again was pioneered many, many years ago, but was recently brought to light again by Dr. Redman in Oxford, is the determination of a serum uric acid. If one plots out a three-dimensional graph showing plasma uric acid levels from 3-8 mg/dl to weeks of gestation, 22-42 against percentage perinatal deaths, you can see there is a very obvious cut-off about 6 mg%, and there is a superb correlation between perinatal deaths per se, degree of pre-eclampsia and the actual level of the plasma urate. This is true at each period of gestation.

Amniocentesis

Amniocentesis too is a very valuable tool that really has not changed too much over the years. The technique of amniocentesis has been improved by the addition of using real-time ultrasound to select the site of the amniocentesis, i.e., to try and stay away from the placenta, so that we do not get significant fetal-maternal bleeds. These amniocenteses can be done at various time-periods. In the very early part of gestation, the so-called genetic amniocenteses are done between 16 and 19 weeks of gestation. There, you are primarily looking for chromosomal abnormalities, neural tube defects and inborn errors of metabolism. Again, it is a service that is offered in most regional centres, and can be very useful in selected cases, particularly in those patients where you think because of maternal age, family history or past obstetrical performance that there may be a particularly high risk of Mongolism or congenital anomalies such as anencephaly.

We can also use anmiocentesis to assess fetal health during the course of the present pregnancy, for example, in Rh disease. We are presently investigating the relationship between the values of amniotic fluid glucose and insulin with subsequent fetal outcome in diabetic pregnancies. Finally, it has been suggested in the past that the presence of meconium in the amniotic fluid, particularly in the post-dates pregnancy, should be an indication for induction. However, recent thinking is that the presence of meconium in the post-dates pregnancy merely represents the last or final stage in fetal maturation, and in itself is not pathologic.

The most common indication for amniocentesis now is to determine the presence or absence of fetal lung maturity. Most laboratories still do L/S ratios, and in the tertiary units, a better or more discriminant test is the determination of diphosphatidyl glycerol. This is particularly beneficial should there be any contamination of the fluid sample with vaginal secretions, blood, or meconium.

With regard to the antenatal detection of neural tube defects in the fetus, I would like to first mention the use of maternal serum alpha fetoprotein. We are presently investigating in Canada whether or not this could be instituted as a routine screening test for all pregnant mothers in the latter part of the first trimester. The validity of such an approach has been proven in areas of high incidence. At present, we are still restricted to the determination of amniotic fluid alpha fetoprotein. An even better test would be amniotic fluid acetyl cholinesterase or synaptic membrane D2 protein. These two tests are presently under investigation and may well replace AFP because they have not been associated with any false positives due to contamination of the amniotic fluid sample with fetal blood.

Finally, a word or two on fetoscopy. It is possible to insert a small fibreoptic system that will fit down the equivalent of a 14 gauge needle, through the uterus, into the amniotic cavity. One can then directly observe the fetus between 15 and 19 weeks of gestation, and, if desired and if conditions permit, take microsamples of fetal blood. This test has proven of major interest in the various blood dyscrasias, particularly Sickle Cell Disease and Thalassemias. These disorders are uncommon in Western Canada, and the test is rarely, if ever, indicated. The test and the laboratory expertise necessary to handle the microsamples is so unique, there are probably no more than four centres in the country which will offer this.

Estrogens

For many years, we have tried to determine fetal condition on the basis of 24 hour collections of maternal urine with estriol assay, or maternal serum estriols. Unfortunately, although these tests may be used for screening purposes, they are not sufficiently sensitive to warn us when a pregnancy should be terminated because of impending fetal compromise. Personally, for this reason, I do not use the test in the Perinatal Unit, but rely instead upon the biophysical testing I will describe later. If and when a reasonable assay for plasma free estriol becomes generally available, this will probably then be the most simple blood test for assessment of fetal well-being in problem pregnancies, if other methods - electronic fetal monitoring or ultrasound - are not immediately available.

ELECTRONIC FETAL MONITORING

In 1821, we have the first record of a physician auscultating the fetal heart rate to determine viability. Later, a Dr. Pryor discovered the association between fetal compromise and bradycardia. By 1947, Barcroft commented upon the relationship between cord occlusion and fetal heart rate decelerations. In 1954, Nicholas Eastman demonstrated the relationship between abnormal fetal heart rate and the subsequent development of cerebral palsy and birth asphyxia. Several studies have shown that clinical auscultation is fraught with counting errors, and, therefore, electronic fetal monitoring is far more accurate. By the early 1960s, there was an incredible boom in the commercial development of electronic fetal monitors which had an immediate, profound impact on the practice of obstetrics and gynaecology. The fetal heart can now be heard using the doppler ultrasound as early as eight and one half to nine weeks of gestation. If the mother can actually hear the fetal heart rate at this very early stage of gestation, it is of far more meaning and significance to her than being told that her uterus is appropriate for dates, or that the pregnancy test is positive. This is particularly so, should there have been any vaginal bleeding and concern about a threatened abortion. Real-time and compound ultrascan also gives remarkably detailed information about early pregnancy. A decidual echo can be seen at five weeks of gestation. By six weeks, there is a 1 cm gestational sac. Within the sac at six and one half weeks, one can find a fetal heart. By seven weeks, the sac is 2 cm in diameter, and by eight weeks, sufficient fetal detail can be determined to visualize not only fetal activity, but also to measure the crown-rump length. For dating purposes, crown-rump length between 8 and 14 weeks is accurate within ±5 days of the true due date. From 12 to 26 weeks, the biparietal diameter will give similar information about pregnancy dating.

Beyond 27 weeks, we again use doppler ultrasound to record trends of fetal heart rate, and determine fetal viability. We rarely do this prior to 27 weeks, because we are rarely contemplating delivery of a child weighing less than 1 kilo. Ultrasound-derived fetal heart rates are useful for non-stress testing, oxytocin challenge testing, as well as the determination of fetal health during labour. Finally, by combining real-time ultrasound imaging with a non-stress test, a full biophysical profile, which includes an objective assessment of both fetal activity and fetal measurements, will provide the ultimate assessment of fetal growth, development and health.

Despite the popularity of fetal heart rate monitoring, the validity of the technique as a predictor of pregnancy outcome was rarely, if ever, subjected to a prospective, randomized and controlled trial. The majority of clinicians, well-versed in the technique, have always considered the information to be crucial to the management, particularly, of high risk pregnancies. Some of us remained skeptical about the benefits, particularly if inappropriate interpretation resulted in an increased Caesarean section rate for so-called fetal distress. Personally, I believe now that there is sufficient data in the literature to suggest that fetal monitoring is of great benefit in high risk pregnancies, although there is no indication to suggest that it should be used as a routine tool in every labour. As the clinician gains familiarity with the technique, and refines his criteria for interpretation, the occurrence of inappropriate Caesarean sections should disappear. There is no good evidence to suggest that invasive monitoring increases puerperal infection or causes any significant increase in fetal infections. Finally, of course, there should be no intrapartum stillbirths in any patient who is being monitored. One problem of interpretation is the difficulty we have in determining the child's sleep status, as Dr. Nathanielsz discusses. There is a distinct periodicity in fetal waking states and sleeping states. One of our signs of fetal well-being is the so-called reactive trace (i.e., fetal heart rate accelerations in response to spontaneous fetal movement). If the child is asleep, it will, of course, not be moving, and the trace will be interpreted as non-reactive. For this reason, technique is most important, and the non-stress test should be prolonged for at least 40 minutes, or possibly one hour to be sure that it is truly non-reactive.

Fetal Movement

The determination of fetal activity is an excellent indicator of fetal well-being. If the child is alive and well, it will periodically be most active, usually just after meals, and between ten o'clock at night and two o'clock in the morning. All one has to do is talk to the mother to confirm this. Such maternal assessments, however, are highly subjective, and we do now have the equipment to perform an objective evaluation of fetal activity using real-time ultrasound. It is fascinating when you watch a mother and show her with real-time ultrasound her child hiccoughing. Often she has felt those movements going on for 10, 15, 20 minutes at a time, every day for the latter part of the pregnancy. She never really knew what it was. I think this is one more indication of the improvement in bonding that can occur if equipment is used properly; it makes the pregnancy far more personal, not only to the mother but also to the husband, who should also be in attendance, if possible. In other words, the different types of electronic fetal monitoring are not intrusive, but can actually be beneficial in enhancing the interpersonal relationships between the mother, father and fetus.

If one considers the fetal movements over the course of gestation, one can identify the onset of fetal activity much as one plots out developmental milestones in the infant. You can see a twitch of the whole fetal body around six to seven weeks of gestation; independent limb movements start at around nine weeks. Isolated head movements are described at ten weeks of gestation, and a combination of arm and head movements occurs shortly thereafter. Diaphragmatic activity can be seen as early as 14-15 weeks, although good, regular respiratory movements involving the whole chest do not occur until 35 weeks. Prior to this time, breathing activity is periodic and irregular. For an objective assessment of fetal activity, we examine the pregnancy for 30 minutes, using real-time ultrasound. We measure five different parameters - fetal breathing, fetal movement, fetal muscle tone, fetal heart rate response to spontaneous fetal activity and the volume of amniotic fluid. Since we score each of these five parameters from zero to two, the total score, or the maximum score that the fetus may obtain is ten. In this respect, the test is very similar to an Apgar. Furthermore, any score equal to or greater than seven indicates a healthy fetus. Dr. Frank Manning, who is presently in Winnipeg, clearly demonstrated a correlation between monitored fetal distress, birth asphyxia, perinatal mortality and the total biophysical score.

Our present regimen in taking care of patients in whom we think there may be some kind of fetal compromise is as follows. In those patients with certain dates and an appropriate size for gestation age fundal height, we initiate the investigation by doing a simple non-stress test. Should this prove to be normal, we would suggest that the patient return in one week's time for reassessment. If, on the other hand, you have any question about the patient's dates, or if the patient's size is inappropriate for her dates, or we have an abnormal non-stress test, we should then proceed on to a full biophysical profile. The non-stress test takes only 20 minutes. A full biophysical profile takes approximately one hour. In those pregnancies where there is evidence of intrauterine growth retardation or placental insufficiency, we would then normally recommend an oxytocin challenge test. The non-stress test and biophysical profiles examine the child under normal environmental conditions, without the intermittent hypoxemic episodes that accompany regular uterine contractions. Many fetuses, even in the presence of an impaired oxygen supply, will adjust their movements accordingly to reduce their oxygen requirement, and it is only by mimicking normal labour by producing three uterine contractions over 10 minutes that we can stress them sufficiently to check the hypoxic fetal heart rate changes. Should the OCT be equivocal or positive, we then will consider the advisability of an immediate induction.

The interpretation of fetal heart rate traces during labour is fraught with problems. Few people will argue with the interpretation of a trace demonstrating recurrent late decelerations. Furthermore, a complicated cord pattern, with baseline tachycardia and loss of variability, is often associated with significant asphyxia. Most patterns will not be consistent with either one of these two diagnoses, and yet they are also not normal. Since we know that the reported false positive, or abnormal, rate of intrapartum traces varies between 50 and 80%, then we have to decide if any other determinant of fetal well-being could differentiate the normal from the abnormal outcome. Failing this, there is always the potential for doing an unnecessary Caesarean section, and then delivering an infant with normal Apgars.

Our first attempt at avoiding a false interpretation of fetal monitor traces was to develop objective scores. We personally use a Kubli score which assesses five different parameters of the fetal heart rate trace, and can be interpreted with a score of 0-10. The five different parameters are baseline heart rate, variability in cycles per minute, amplitude in cycles per minute and beats per minute, accelerations and decelerations. We are attempting a further refinement of this Kubli scoring by trying to feed the output from the fetal monitor directly into a desk-top computer, which will then give us some on-going real-time Kubli analysis of the fetal heart rate trace. No one has yet developed the satisfactory software for the computer, and so this remains an on-going research problem at present. Should we prove successful, however, then the error of subjective assessment should be eliminated.

The most recent refinements in the technique of fetal monitoring involve intermittent fetal scalp sampling and continuous transcutaneous determinations of fetal scalp gases. For years, we had the ability to draw fetal scalp blood for assessment of pH and blood gases. Technically, however, this was not a particularly easy test to perform, and, despite the initial flurry of activity in the 1960s, even most perinatal units have now abandoned the technique, except in unusual circumstances. In intensive care units over the past several years, they have had transcutaneous sensors capable of detecting tissue pO_2 and pCO_2. Furthermore, it has become apparent that these transcutaneous values are extremely labile, and, for this reason, intermittent sampling may not be representative of the underlying clinical condition. In the neonate, for instance, measured pO_2 values will fall during suctioning or any other form of instrumentation, and therefore, samples drawn during this temporary reduction will be totally unrepresentative of the underlying core values. Dr. Nathanielsz has already shown the effects of myometrial contractions on the intervillous space blood flow. Since the fetus is totally dependent upon the oxygen content of that blood in the intervillous space, it is not surprising that there is a delayed period or lag period of approximately 40-50 seconds from the peak of the contraction to the low point in the fetal oxygenation. At present, we are in the process of studying the correlations between uterine contractions, fetal heart rate trend and transcutaneous blood gases. We are particularly interested in the transitions that occur with the onset of respirations at birth.

SUMMARY

My first recommendation to anyone concerned about the evaluation of a high risk pregnancy is to deliberately take the time to get a detailed history from the patient. This, coupled with a pertinent physical examination using objective assessments of uterine size, e.g., symphysis-fundal height, will lead to early diagnosis of most problems with fetal growth. Anticipation of pre-eclampsia can be refined by means of use of blood uric acid evaluations and the provocative rollover test. Judicious use of ultrasound, both real-time and compound, will detect early fetal viability, confirm dates, measure growth, and identify many fetal anomalies. Subjective and objective assessment of fetal activity will give us a good evaluation of fetal health. In selected cases, an amniocentesis will provide much-needed data on chromosomal aberrations, neural tube defects and inborn errors of metabolism. Furthermore, it may give us vital information on fetal health in such disorders as erythroblastosis and perhaps diabetes. Most commonly, of course, it will confirm the fetal maturity in those cases that have questionable dates and no early ultrasound. Electronic fetal monitoring has now come of age, and, with objective assessment of antenatal testing and careful evaluation of intrapartum traces, supplemented, if necessary, with intermittent scalp sampling or possibly continuous trend pO_2 and pCO_2, we should be able to diagnose asphyxia early. Early quantitation and deliberate shortening of the duration of asphyxia by judicious use of Caesarean section and/or forceps delivery will improve neonatal outcome.

Finally, I would re-emphasize that in obstetrics, we are dealing with a patient and a family, and not just a disease process. Listen to the patient. Do not be afraid to "lay on hands" and carefully evaluate uterine growth. If ancillary aids are to be used, explain their purpose to the patient. Encourage the patient's husband to be present wherever possible during the antenatal fetal heart assessment, or real-time scanning, so that he too can participate in the excitement of the moment. I would consider this to be just as important as attendance at prenatal classes, and hopefully, his attendance at labour and delivery. Even fetal monitoring in labour does not have to be obtrusive. The newer fetal monitors can work equally well on the basis of telemetered data, and, therefore, the patient can be up and about as much as she wishes during the course of labour. The better educated the public is, the more willing they are to co-operate during our attempts to obtain a healthy baby, and healthy parents following delivery.

RISK SCORE

(INCREASE IN PERINATAL MORTALITY)

★ Red	Gestation 31 weeks Placental insufficiency Hydramnios (single fetus) Diabetes (insulin) Bleeding Twins	x10
★ Yellow	Erythroblastosis Gestation 32-35 weeks Pre-eclampsia (BP 140/90 & over + protein) Breech	x5
★ Blue	Single parent Age 35 years and over	x2

★ Green

A) Obstetric Factors

- Postmaturity (42+ weeks)
- Prolonged rupture of membranes (24+ hrs)
- Previous cervical trauma
- Previous Cesarean section
- Fever and/or amnionitis

B) Medical Problems

- Renal disease
- Thyroid disease
- Lupus
- Crohn's disease & ulcerative colitis
- Infectious hepatitis
- Addicted patient

NB Incomplete data but significant high-risk factors

E.F.M.

HISTORICAL DEVELOPMENT

1821	De Kergaradic - auscultation fetal heart
1851	Preyer - bradycardia, placental insufficiency
1947	Barcroft - deceleration, cord occlusion
1954	Eastman - 50% of CP, perinatal asphyxia
1957	Hon, Hess - errors of auscultation
1960's	Commercial development

ANTENATAL SCREENING E.F.M.

I	FETAL HEART DETECTORS U/S	9 weeks
II	REAL-TIME & COMPOUND U/S	6 weeks
III	FETAL HEART RATE TESTING Doppler U/S Abdominal FECG Phonocardiography eg NST, OCT (CST)	27 weeks
IV	COMBINATIONS eg BPP	

LITERATURE - MONITORING IN LABOUR

A **Retrospective**

99% of literature

B **Uncontrolled Prospective**

Paul et al 1977

Neutra et al 1978

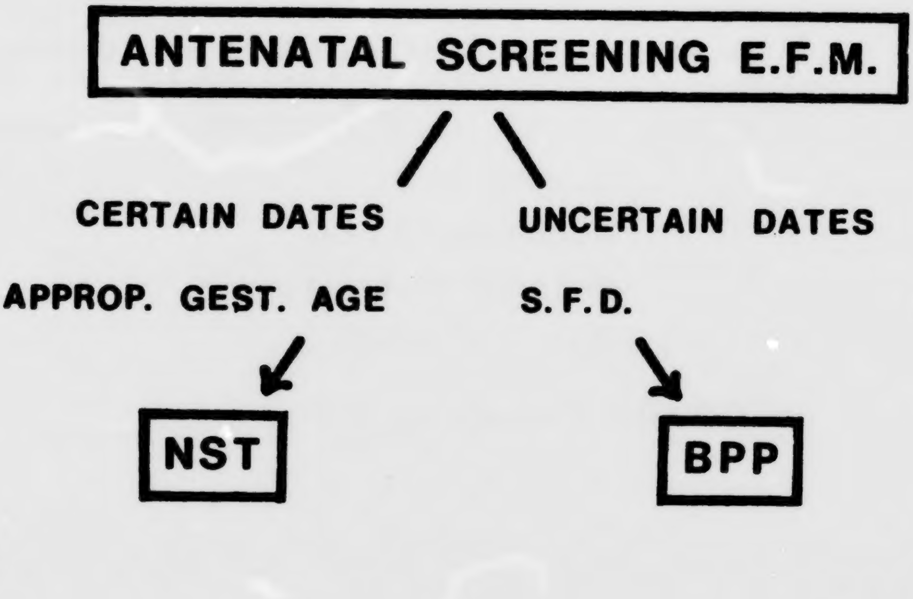
C **Randomized Controlled Trials**

Hi-risk

- Langendoerfer et al 1980
- Haverkamp et al 1979
- Haverkamp et al 1976
- Renou et al 1976

Lo-risk

- Kelso et al 1978



ANTENATAL SCREENING E.F.M.

CERTAIN DATES

APPROP. GEST. AGE

NST

UNCERTAIN DATES

S.F.D.

BPP

E. F. M.

RISKS	BENEFITS	
Uterine perforation	Avoid supine position	↓ C.S.
Scalp infection-3/1000	Ambulate	↑ Apgar (but)
Amnionitis	Bonding	↓ PNM (but)
↑ C.S. (fetal distress)	Nursing efficiency	
	Detection of fetal compromize before labour (NST, CST, BPP)	
	Prolongation of pregnancy in diabetes, etc.	

ASSESSMENT FOR SENSORI-MOTOR STIMULATION

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Before discussing "Assessment For Sensori-motor Stimulation", it is essential that these terms be defined, if they are not to be misunderstood. I would like to quote from the recent review by Ferry(1) to illustrate the extravagance of confusion resulting from failure to define "stimulation".

> "Premature infants...are being cuddled, patted, stroked with vibrators and rocked in waterbeds and motorised hammocks. They are exposed to flashing lights, dangling birds and toys, and piped maternal heart-beats and music. Infants and preschoolers with neurological disorders roll on beachballs. Older children with learning disorders practise walking on balance beams to improve their gross motor skills and learning abilities. 'Infant Stim' and development therapy programs now represent a major industry."

The word 'Stimulation', whilst grossly inappropriate in the light of our knowledge of the transactional basis of human development, is here to stay. In the transactional view, the developing organism is an active participant in its own progress, which it effects by imposing change upon its total environment and reacting to the new situation it has created by some form of learning, consolidation, or other behaviour. 'Stimulation', in the dictionary sense of 'excitation to increased activity', is often the last thing required by the developing infant. The definition I would offer to those seeking to benefit the developmentally 'at-risk' says:

> Stimulation is the provision of appropriate and rewarding opportunities for learning through action and transaction with a human caretaker, such that motor and other responses are perceived as worthwhile.

This definition implies that learning, or progress through any sort of developmental agenda, must be perceived as worthwhile to the individual, and implants right from the start the ideas of self-worth, and experience of success. By stressing the word 'appropriate', we recognize that each infant has unique needs, and by mentioning 'opportunities', we acknowledge that the infant, rather than the caretaker, will often determine the moment for and duration of the learning experience. Such a definition dictates the type of assessment to be done.

Ferry,(1) in her already quoted critique of the 'Infant Stim' movement, concluded "there is no valid evidence that these programs do alter neurologic development in high-risk or neurologically handicapped children". Unfortunately, this statement as it stands is true. There is no evidence that damage can be undone by external influence nor that such limited capacity for natural repair as exists within the central nervous system can be enhanced by input.(2) The burning question is, to what extent are the most significant features of human development 'neurological'? If two individuals with identical neurological deficits have outcomes which are recognizably different in the real world, then influences other than those of the deficit must have been present.

Socio-economic status has repeatedly been shown to be a more reliable predictor of developmental outcome than any biological factor, its influence amplifying, reducing or just outweighing the effects of perinatal injury or low birth weight.(3) Neligan, et al.,(4) demonstrated that the socio-economic status effects were largely mediated through observable features of maternal care and expectations, to the extent that paternal occupation could be virtually eliminated as a statistical power on outcome. The concept that low birthweight infants, and light-for-dates infants in particular, show an excessive vulnerability to adverse psychosocial factors appears valid. Whilst intrauterine growth impairment especially, and low birthweight and perinatal stress in general, may produce widespread and non-specific impairment of school-age performance, Neligan's(4) epidemiologically-based longitudinal survey revealed how trivial their effects are in comparison to simple "quality of parenting" variables. Understanding of these mechanisms has been greatly enhanced by use of standardized environmental process measures.(5)

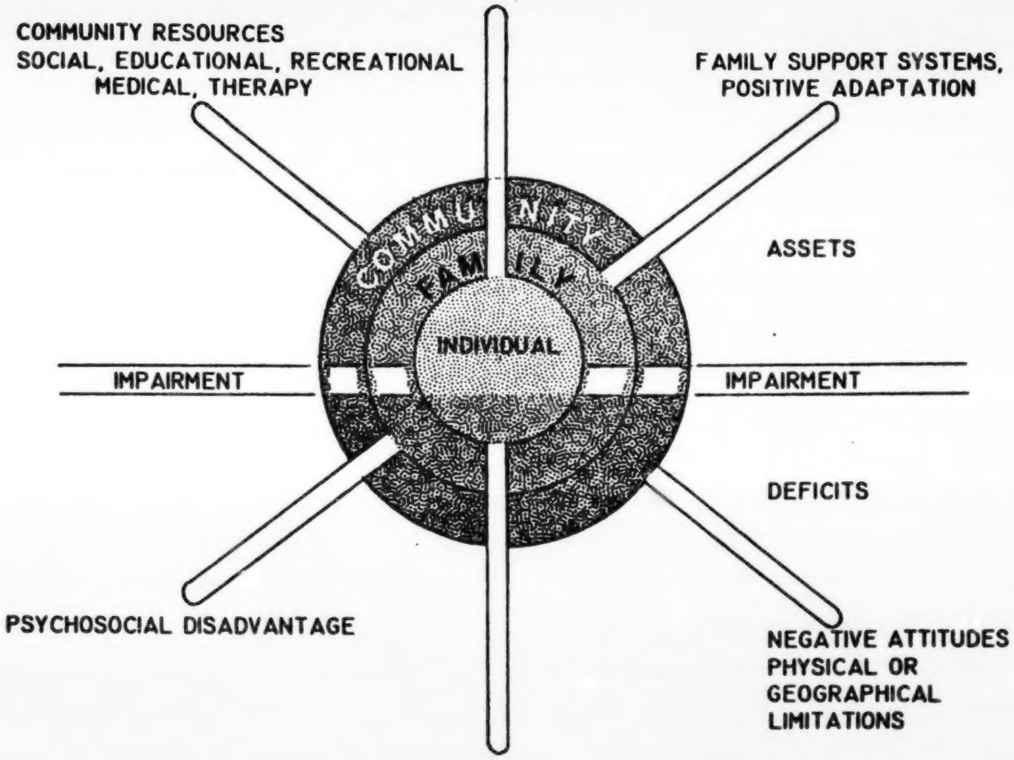
What is distinct about the first two years is that it is only during this period that, in general, the biologic or neurologic influences upon high-risk groups of children seem to outweigh the social factors. The later, after the second birthday, that we look for group sequelae of obstetric factors such as asphyxia or rooming-in, or medical conditions such as bronchopulmonary dysplasia or hypertonicity, the harder it is to find positive correlations, even though in the first two years all these have powerful and easily discernible effects upon development as usually assessed.

This is only another way of stating the general rule that in human development, the power of a given influence will reflect its intensity and the duration over which it operates rather than the time at which it is applied, given a responsive biological substrate on which to work. This applies to asphyxia or immobility as much as to good or poor parenting or 'stimulation'. The sensori-motor period could be redefined as that time when the measured effects of biological or pathological variables still outweigh those of interpersonal or sociological factors. Paradoxically, however, it is just because we cannot, to any great extent, alter the effects of biological deficit or pathological injury that we are left with an increasing possibility of conferring benefit, if the environment can be made optimally responsive to the needs of the individual child. By inducing changes in the environment which are either permanent or of long duration, and which are in the direction of providing positive responses contingent upon behaviours which are socially desirable, we can promote transition from one level of developmental activity to another, and most important confer upon the child experience and expectation of success, pride in achievement and drive toward mastery. Stimulation in the sensori-motor period offers the possibility of setting up habits of reinforcement and adaptation which might continue to facilitate optimal development long after, far outweighing the observed benefit, or escaping our means of evaluation, during the limited period of actual programming.

The dictionary definition of assessment reminds us of some etymological roots which are of immense clinical significance. Assessment implies the evaluation of assets, with overtones of "sitting down together" or "putting one's self in another's shoes". How often does assessment become an occasion for identifying deficits and inhibiting empathy with infant or parent? The term is often wrongly used to describe more or less subjective testing along one or more dimensions. Because of the complexity of human development - an area in which each new discovery leaves us more aware of the extent of our ignorance - developmental assessment must be carefully distinguished from the simple developmental "impression" embodied in clinical observation or history-taking, or the utilization of screening tests or inventories or the more complex, but still one dimensional examination performed using a standardized situation or techniques by a trained professional. One person cannot perform an assessment simply because no one person can identify all the assets and deficits of a developmental situation. That is one indication for multidisciplinary assessment; another exists whenever there is a problem to which there is more than one possible solution. So, when we talk of developmental assessment, we must mean a 'comprehensive' assessment, concerned with at least three levels of organization and with the interfaces between them. Assessment must acknowledge what has been learned from the study of child development and must relate to the theoretical basis of whatever interventions may or may not be recommended.

The first level is that of the individual - the infant - being assessed, and involves insight into his biological strengths and weaknesses as well as into his personal uniqueness and knowledge of his progress in adaptive, integrative, affective and cognitive areas. One must know not only what the infant does and does not do, but who the infant is and why he shows given abilities or limitations, where he is coming from and when he arrived at his present level of function.

The second level is that of the family, and minimal requirements include knowledge of the child-rearing patterns available as models to both parents, of expectations derived from each or any prior experience of either parenting or infant behaviour, or inter-personal relations, of protective factors and of present or past stresses whether economic, social or emotional. Assets are to be identified as rigorously as liabilities.


COMMUNITY RESOURCES
SOCIAL, EDUCATIONAL, RECREATIONAL
MEDICAL, THERAPY
FAMILY SUPPORT SYSTEMS,
POSITIVE ADAPTATION
COMMUNITY
FAMILY
INDIVIDUAL
ASSETS
IMPAIRMENT
IMPAIRMENT
DEFICITS
PSYCHOSOCIAL DISADVANTAGE
NEGATIVE ATTITUDES
PHYSICAL OR
GEOGRAPHICAL
LIMITATIONS

The third level of analysis is that of the Community in which the family functions, its expectations and attitudes, geographical and climatic factors, and its human and technical resources. Where cultural factors operate to alter developmental expectations or norms, or provide unexpected supportive factors, these too must be evaluated. Particular attention must be given to the interfaces between two levels - the effect of the child on the parents and the family on the child, or the outer and inner directedness of the family in regard to the caring or uncaring outside world.

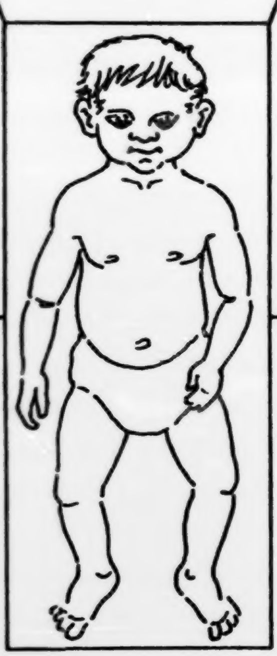
Each of these multifaceted and complex levels must be integrated to provide a basis for the first stage of what will be recognized as a process of assessment. Whether the assessment be conducted by the minimum core team of social worker, physician and psychologist or by some equivalent core team, or by an augmented team including a variety of educators, therapists, or other professionals, each will go through a sequence of acquisition, verification and integration of data to furnish a description at one or more levels. The 'description' provided by any one professional is likely to reflect the training and perspectives of one discipline, and each must be integrated in the process of assessment to provide a unified and mutually acceptable formulation. In the penultimate phase of comprehensive assessment, this integrated formulation is used as a basis for 'prescription' - the recommendation and implementation of specific intervention measures, negotiated between and acceptable to all the professional viewpoints and, essentially, the parents. The last phase of assessment is 're-assessment' - because time is a cardinal dimension both in human development and in its assessment, and assessment works best when observations of change over time, with and without intervention, are incorporated.

Assessment, then, may be defined as "a process, in which information gathered over as wide a base as possible is integrated with current neurodevelopmental theory to produce agreed and negotiated consensus formulation, sensitive to the requirements and viewpoints of parents and of a variety of professional disciplines, which serves as a rational basis for the deployment or withholding of community resources and for the design and implementation of programmed environmental change according to the needs of the child

WHO is he?

How is this child unique?

Temperament
Style



WHERE
is he coming from?

Biography
Prior documentation
Previous experience
Obstetric/perinatal
Nutrition

WHAT he does?
Abilities and limitations

Organisation of Self and Society
Interpersonal Relations
Communication
Control of environment:
self-help and fine motor
Control of space:
gross motor

WHEN

did he reach present
level of function?

Rate of progress
within and between
developmental levels

WHY he shows +ve and
-ve features:

genetics
social environment
pathophysiology
morbid anatomy
sensory or motor deficits
protective or facilitating factors

assessed in relation to his disability, handicap, risk-status, development, family and society."

Such comprehensive developmental assessments might appear too complex or too expensive for the practical needs of 'Infant Stim' - yet, without sophisticated assessment, prescriptions are likely to be inappropriate, incomplete, and ineffective; programs are expensive, and divert highly motivated and often elaborately trained personnel from involvement in comparable tasks whose benefit is better substantiated. Furthermore, 'Infant Stim' is not without dangers: there is a real risk of professionalizing the art of parenting, of creating unnecessary dependency, of producing in infancy and early childhood the cruel pressures of conformity and negative labelling which stifle the individuality and confidence of all too many school children who meet head on an inflexible system of norm-based expectations. If the procedures employed, correctly applied, can do good, then their inappropriate use is potentially harmful. If limited resources are to be deployed in the most effective way, it is imperative that Assessment for Sensori-motor Stimulation be capable of recognizing those situations in which no intervention is going to influence or where outcome will be as good without help; the type of intervention appropriate to the particular situation must be individualized, and identification made of all factors, positive and negative, which may be active in a given case. As a physician, one is much aware of the futility of programs which fail to recognize the delaying effect of physical illness or the apparent developmental acceleration that spontaneously follows acquisition of good health. Programs must be tolerant of the multitude of varieties of effective parenting behaviour, for just as individual children may have contrasting but equally productive behavioural or learning styles, so may superficially divergent approaches to parenting have equally adaptive results.

Thus, in addition to its child and family focused information-gathering and prescribing functions, the assessment process must act to support parents in their role of primary caretakers and must be so structured as to encourage their active participation in, and responsibility for, decision making. Whilst most parents in high-risk groups welcome surveillance and, if necessary, programming, the assessment process must recognize the need or right of some to go their own way, must avoid both constraining families into

COMMUNITY ANALYSIS

1. Attitudes - institutionalised power
   - non-institutionalised influence
2. Geographical limitations and relationships
3. Economic pressures and resources
4. Institutionalised resources
   - Medical Public Health, Nursing
     - Obstetric, paediatric, psychiatric
   - Mental Health Centres
   - Habilitation/Rehabilitation professionals
   - Social Service Clubs
     - Voluntary Societies
     - Ethnic associations
     - Churches
     - Childrens Aid Societies
   - Education School Board, Teachers
     - Day Care agencies, staff
5. Non-institutionalised resources
   - Parent groups, Parent participating schools, Latent expertise

TASKS FOR THE ASSESSMENT PROCESS

1. To support Parents in their role of primary caretakers, encouraging their active participation in and responsibility for Decision-making.

2. To provide as full a Description of the child, in the context of the family and community, as is required through identification of Strengths and Deficits to develop a total Prescription for intervention and/or surveillance.

3. To set goals such that the impact of programming on the individual child and family can be monitored.

4. To provide a basis for Program Evaluation.

compliance, and sowing seeds of guilt for independence. Professionals must avoid making unrealistic claims and must use communication skills of high order to present themselves as humanly caring and helpful rather than authoritarian, expert or essential. Lastly, the assessment process must set measurable goals by which the progress of the individual child, and the efficacy of the entire program, may alike be evaluated.

I will now review some details of the assessment process, working in from the community through the family and parenting to the description of the strengths and deficits of the child.

There is no need to enlarge on the difference between helping programs delivered by intimates directly into the home or within the neighbourhood and those provided by more or less remote professionals working from a central agency. Similarly no discussion is required on the contrast between cohesive mutually supportive and self-sufficient communities and those which are anonymous, outer directed and dependent. No assessment, however, can afford to proceed as though these variables were unimportant, and no assessment team can fulfill its purpose without access to some form of community analysis or inside knowledge. Minimal requirements for community analysis include some indications of prevailing attitudes within both institutionalized and non-institutionalized sources of power and influence, awareness of the impact of climate and geography, and reasonable familiarity with the economy of the area. The assessment team must be aware of all the institutionalized resources which can be deployed, now and in the future, to further its goals and participate in problem solving. Much can be achieved with parent groups, and the insider will sometimes recognize that the ideal solution to the difficult problem is already at hand. Much information of this type is susceptible to computerization, but such data banks do require constant updating and re-evaluation.

Community factors impose constraints upon professionals and families alike. Family function in the widest sense cannot be ignored in any meaningful assessment, and, while the particular bailiwick of the social worker, it demands some inquiry and concern from physician, psychologist or any developmental therapist. In its effect upon outcome of high-risk or damaged infants, parenting is the cardinal aspect of family function to be addressed, but parenting itself reflects other family activities. We lack effective measures of parenting, and, in particular, we lack evaluations which are class and culture free and incorporate theoretical advances in the understanding of parenting derived from objective laboratory observations. We are beginning, however, to have reasonably standardized ways of recognizing a "good home". While social class is a powerful predictor of many kinds of group outcome, and is increasingly recognized as such in obstetric-perinatal outcome studies, it is unsafe and unhelpful in many specific instances. Measurements based on the acquisition of status, whether of income, education, housing or employment, neither predict nor explain intra-class variation nor do they allow the identification and reinforcement of protective factors which undoubtedly exist. The need for process-related ratings of the learning environment has been long recognized and such instruments are demonstrably superior to status measurements of environmental quality.(6)

The HOME (Home Observation for Measurement of the Environment),(7) has acceptable psychometric and epidemiologic characteristics for employment as a screening tool. It permits one to recognize the existence of protective factors and to allow for these in evaluation of results or in deploying scant resources. HOME has been found a good predictor of the development of premature infants, and it is of interest that among the premature the relative potency of the various sub-scales as predictors of cognitive performance differs from that generally found: thus, avoidance of restriction and punishment may be especially important in this group. It takes less than one week's training to achieve high inter-coder reliability. It is administered in the home at a convenient time when the child is present. It has been used effectively to predict mental retardation, language handicap and malnutrition. Scores on the HOME correlate with later I.Q. better than $r = 0.5$ and r values of 0.7 are found in some items. Several studies(5,8) have found high correlations between Bayley scores and measures of parental involvement, provision of appropriate play material or "variety of stimulus input". The provision of an increasingly varied and, within certain limits, "surprising" set of stimulating circumstances is, together with their greater verbal interaction and encouragement of mastery, perhaps the key attribute found in parents of "competent" children. Parental behaviours which increase the child's conviction that he is loved and valued are amongst those factors which, when present, tend to diminish the legacy of early biological insult, and which must be identified and accentuated by any assessment process or therapeutic intervention. Such behaviours are probably identical with those required to facilitate the emergence of what Burton White(9) called "competence". Competent children have the ability to engage and maintain the attention of adults in socially acceptable ways and to use them as resources; to express both hostility and affection to adults and peers; lead, follow and compete with peers; and imitate adult roles. In the concept of "competence" both social and cognitive proficiencies appear to meet: and it looks as though the same type of parental support is required for both components.

Here the concept of "match" and "mismatch" between parent and child assumes importance. Effective parents know that one cannot "treat all children alike" and quickly learn how "this child is different from all other children". The ability of parents to modify their own behaviour according to the requirements of the individual child becomes crucial. The converse of White's definitions of childhood competence demands parents who are prepared and able to have their attention engaged and maintained by their offspring, to be used as resources, and to be the recipients of hostility as well as of affection. Fox(10) defined parenting as "the ability of mothers and fathers to understand, anticipate and make mutually satisfactory adaptations to the individual personality and needs of their offspring so as to enhance achievement of developmental goals".

Although one can lay down some broad strategic goals for parenting behaviour, one cannot define the precise way in which those goals will be met. There are as many ways of demonstrating positive involvement with one's children, encouraging mastery, or allowing oneself to be used as a resource as there are temperamental variations amongst infants; there are as many ways of indicating to an infant that he is loved and valued, which emerges as a crucially predictive developmental parameter, as there are of composing an amatory sonnet. As Kagan(11) stressed, "Parental love is a belief held by the child, not a set of actions by a parent. The belief that one is valued does not lie in a particular set of parental actions, but in the child's construction of those actions."

The pre-eminence of process over practice as the determinant of outcome needs to be stressed. Therapists and assessment teams must have an eclectic and open-minded approach to the mechanics of childrearing, since there is no evidence to show that any specific infant care techniques have any unvarying psychological effects upon children in later years,(12) perhaps because "one and the same practice may be performed by a nurturant or a hostile mother, may occur within an easygoing or a rigidly authoritarian home, or may take place against a background of love or hate." Assessment tools are required, which are able to identify situations where process and relationships are likely to be maladaptive, and equally where protective factors might reasonably and safely be expected to obtain. Ideally we require instruments which are not only predictive, but provide a rationale for specific intervention techniques.

Parenting quality may be assessed by attempting to match parental perceptions of the needs of the child against independent norms, and by observation of parent-child interactions. It cannot be pretended that clinical procedures, employed by concerned practitioners, approach the validity of biochemical tests. However, Broussard and Hartner(13) were able to predict outcome significantly in a series of 120 neonates reviewed at four and a half years on the basis of a simple questionnaire on mothers' perceptions of their infants. Prediction here was high enough to suggest employment of this measure to indicate the need for preventive intervention. This study has been continued up to eleven years with consistent results (unpublished).

The clinical evaluation of parenting involves observation of parent-child interactions. Affective display during nurturing tasks provides important clues to the individual relationship(14) in terms of enjoyment and anxiety. The perceptiveness of the mother regarding the specific needs of the child, and in particular her capacity to respond flexibly and appropriately(15) and without undue deference to her own dependent needs may be informally observed. Simple measures may be highly significant: thus, Levy would invariably tell mothers "That's a very pretty baby" - mothers who did not glance down at the infant were found to show impairment later. Others have made similar observations of duration of maternal regard, encouragement and support during testing or manifest ability to anticipate needs, particularly ability to soothe.

"Conversations" between mother and infant have been extensively studied in the laboratory, and may be witnessed in the office. Utilizing video-analysis of contingent maternal behaviours, Greenspan and Lieberman(16) have developed some empiric measures which may eventually reach field-level usefulness. Quantifiable measures of interaction are desperately needed.

A history of adverse childhood experiences in either parent can be easily and casually obtained in many instances, and alerts the clinician to search for protective factors or problems. Histories of bonding delay or failure ("When did you fall in love with your baby, or when did you feel that he was really yours?") elicit a wealth of useful material. History of the everyday life experience of mother and child may alert one to parenting discrepancies.

The cognitive approach to evaluation of parenting(17) studies the parents' own attempts to make sense of their role and their experience. Such an approach is developmental in that it posits an increasingly complex structure of thinking and allows for transformations from one level to the next through interaction with the environment. Whilst clinical research observations suggest that in younger mothers bonding may be sub-optimal and inconstant(18), that their infants show less mature physiologic and motoric control(19) and that parents who cannot empathize with their children tend to present with childrearing difficulties, the cognitive approach studies the parents' reasoning about their child's experiences, the effects of experience on the child, and their ideas of responsibility in parenting. Succeeding levels of so-called "Parent Awareness" have been delineated, and can be identified in the research interview, ranging from a primitive egotistic orientation in which the child is viewed simply as a projection of the parent, with necessarily congruent needs, through a conventional level of parenting role-organization within the constraints of socially-defined notions of what is right, to levels in which uniqueness of the child is recognized as transcending normative prescriptions and values. At the highest level parents understand their child as a complex and changing psychological system and are able to modify their relationship and role to achieve responsible means of meeting their own needs as well as their child's.

It may well be that what is important in the first two years of life is the development of the caretaker rather than that of the infant. Perhaps during these two years it is the dynamic personal growth of the parent that in essence determines the outcome of the transactional process in which the infant, recovering from, damaged in, or unscathed by, the multiple perinatal hazards of obstetric risk, asphyxia, low birthweight or other insult, is an active participant.

Only now, after considering some approaches to the evaluation of the community, the family and the parents, can we safely look at the assessment of the infant.

Essentially, each discipline involved in a comprehensive assessment will go through a similar procedure to answer, in respect of a range of age-appropriate activities, the questions - is this child, in respect of this function, normal, delayed or deviant? If delayed, what are the consequences of this delay for the parents, for the performance of the child in other areas of development, and for the child in the area of delay? What is the mechanism of the delay? Will it self-correct or can we recommend appropriate interventions? In the case of deviant behaviour, findings which are never seen in a normal child such as seizures, abnormal neurological findings, extreme degrees of delay not accountable for by environmental factors or attributable to prematurity alone, autism, deviant communication patterns and so on, special care will be taken over medical investigation and the approach is more likely to be therapy based and child centred, than a predominantly parent education/infant stimulation approach.

Assessment involves priority ranking of a number of possible interventions on grounds of need, availability, cost effectiveness and so on. A developmental approach is invaluable. Als(20) has provided a conceptual framework of sequential interdependent and expanding levels of infant organization within the infant-caretaker system. At successive levels, infants are faced with a developmental agenda, which must be, to some degree, completed before pro-

Primary Intervention*	Incomplete Agenda	Possible Manifestations
MEDICAL	PHYSIOLOGIC HOMEOSTASIS	Survival; organic injury; impaired transactions
THERAPY	MOTOR CONTROL ↙ ↓ ↘ Feeding Gross Fine	Motor delay; tone distortion; Management problems; impaired transactions
STIMULATION	STATE CONTROL	Attentivity; ? memory Management problems; impaired transactions
PARENT EDUCATION	SOCIAL INTERACTION	Relationship difficulties Deprivation Communication

* considerable overlap and variation

Captive Audience

ceeding into, and enlarging upon, the next stage. Each agenda, however, continues with increasing complexity into later age levels. Thus, at around the 28 weeks level, the agenda comprises physiological homeostasis against either internal or external stresses. The infant who has unfinished business in this area may demonstrate apnea, color changes, or excessive heart-rate liability in the face of inappropriate stimulation for weeks or even months to come. Around 36 weeks gestation the emphasis shifts to increasing motoric differentiation and modulated control of movement patterns; the infant who has incomplete assignments here may have maladaptive hypertonicity or fail to evolve anti-gravity defenses later on. A month or so after term, the ability to generate, maintain, and transfer between arousal or behavioural "states", so as to be available for appropriate stimulation whilst producing adaptive defenses to the inappropriate, is the key activity. Difficulties here may translate into varieties of attention control problems, representing either immaturity or disorder, and might relate to eventual development of memory. By three months, the infant who has done well in these essential underpinnings is ready to utilize physiological homeostasis, motor control, and the ability to deploy arousal states effectively to elicit and increasingly control fruitful social interactions. Here are the seeds of positive responses, drive and self-esteem. Conceptually, it now becomes easy to attach more specific areas of developmental endeavour - gross and fine motor, social adaptive skills, increasingly complex non-verbal communication, symbolization and language - into this framework. Treatment modalities of interventions may be ranked in reference to their central position amongst these expanding concentric cones. It becomes easier to understand why the infant with broncho-pulmonary dysplasia who cannot achieve physiological homeostasis may show no developmental progress for months and may then, with the acquisition of mature lungs, rapidly double or triple his developmental quotient almost spontaneously. Clearly in such a case, the priorities would be firstly life support and avoidance of overwhelming stress, secondly preservation of the family supports in the face of an unresponsive and vulnerable infant, and thirdly perhaps physical therapy to prevent contractures and deformities or the atrophy of sensory end-organs in immobile limbs and joints(21).

Similarly, the child whose movement patterns are dominated by hypertonicity, perhaps due to cerebral palsy, perhaps reflecting a persisting tonic labyrinthine reflex due to faulty perception of the neutral or anatomical position following months hyperextended on the respirator, or perhaps associated with the healing of an asphyxial insult, must either wait for time to resolve the abnormal posturing and remain the while imprisoned within a spastic straight-jacket or receive the specialized attention of the physical or other therapist trained in neurodevelopmental or comparable techniques for tone reduction. So different is the motor development of the infant under 30 weeks gestation that a physical therapy approach is almost mandatory.

Sensori-motor input at any age, if it is to be beneficial, must be appropriate to the receptive capacity of the infant. A premature infant, or one recovering from insult, may respond aversively or protectively to a gentle stimulus such as a soft rattle to which the mature thriving neonate will orient and search. Whilst there are suggestions that stage appropriate input may have beneficial effects upon weight gain and short-term developmental status in the newborn,(22, 23, 24) the over-stimulated infant may fail to learn facilitative patterns of integration and his experiences may contribute to the "irritable baby syndrome", to transient dystonias or to infantile colic with definitely maladaptive consequences for most parents.

The quiet alert state is hard to achieve in not a few newborns, but by four weeks it becomes increasingly available for the ordered receipt of social stimulation(20). Mothers of high-risk infants have been noted(25) to be more intrusive in playing with their four-month babies than the mothers of normals; such intrusiveness is related to their infants' measured relative unresponsiveness at term, and parental sensitivity and ability to avoid intrusive play was predictive of vocabulary and complexity of language production at two years. Field suggests that these mothers of high-risk infants have acquired a particular style of stimulation which, rather than facilitating attentiveness, served to overwhelm the fragile infants, producing further declines in effective receptivity. Whilst the form of intrusiveness changed, the style seems to persist since these mothers were recorded as using more imperatives when playing with these toddlers, and these command frequencies were highly correlated with poor two-year language development and persisting pre-school and possibly later difficulties. This is an excellent example of how complex transactional development is and points to the necessity of individualizing input to needs and not simply to deficits.

Tests and observation procedures must be increasingly attuned to these complexities and must be designed to reveal the uniqueness of each developing organism.

Such an instrument is the Neonatal Behavioural Assessment Scale (NBAS)(26). This emphasizes the infant's stage of arousal and his capacity for adaptive change from one state to another, as well as recording habituation or response decrement, which can surely be regarded as some sort of analogue of early learning. The predictive efficiency of the NBAS, while certainly comparing favourably with standard neurological examinations,(27) is more usefully applied to groups than to individuals. Serial administration reflects the newborn's recovery from the stress of labour, delivery and the impact of environmental change, as well as from injury. The newborn is seen as a recovering organism, and the rate of recovery as the best index of growing capacity to attend to social stimuli(28). The scale is creatively used not simply as an assessment or research tool but as a shared therapeutic experience for parents, who can experience growing awareness of their baby's depth and range of behaviours, and become attuned to the implications of signals which may otherwise be misinterpreted or more simply may not be recognized. Training requirements for users of the NBAS are rigorous and administration is time-consuming, but the therapeutic benefits may justify this. Parents who have participated in the administration of NBAS or in any comparable experience which allows them an informed, empathic commentary on the feelings, capacities and idiosyncracies of their offspring, are rarely left unmoved, but more attuned to the transactional task before them. Indeed, participation in this test has been shown to produce a significant increase in Bayley score at 12 months(29). Infant Stim begins here!

(A modification of the NBAS for the premature is in the research stage,(30) and an abbreviated clinical tool has recently been described by Berger.(31))

Another series of instruments, which investigate qualitative individual differences in a standardized manner, are those measures of temperament deriving ultimately from the work of Thomas and Chess.(32) Each provides profiles of the nine behavioural styles or temperamental traits derived from the New York longitudinal study, which manifest themselves in changing ways with increasing age and demands on the child, but show some stability over the years. Such traits contribute to the transactional process, modifying the organism's reception of and response to environmental stimuli, and in turn eliciting social responses from salient adults which may be more or less adaptive for the child. Traits are regarded as probably genetic in origin, but susceptible to modification by experience, and both in assessment and programming enhance understanding of the "how and why" of development.(33) Parent counselling may focus on the most fruitful adaptations to particular combinations of traits as well as increasing awareness of the child as a unique person. All the temperament questionnaires share internal consistency around 0.85, and the research evidence is that in general they are accurate descriptors of child behaviour rather than simply of maternal perceptions.(34) Clinically, they may be used to give insight into parent/professional or inter-parent discrepancies in perception or expectation.

An astonishing number of tests and inventories is available for the investigation of the "what" of development rather than the "who" or the "how". Perhaps the actual test used is less important than the use which is made of the results, and the extent to which the limits of testing and the place of test-results in the whole context of developmental assessment is understood. Any test should be supplemented by free-field observation, and must be integrated with parental report and with the social, medical and other views of the child.

The distinction between norm referenced and criterion based measures is of some importance.(35) The former compares an individual's performance on a series of identical tasks with that of a peer group, the meaningfulness of the score emerging only on comparison. Instruments such as the Bayley,(36) Knobloch-Gesell,(37) or Griffiths,(38) are of this type, and so in essence are the less rigorously standardized, more clinical tools of Illingworth,(39) Sheridan,(40) or Frankenburg,(41) which are of greater value in the formation of first-level developmental impressions than in assessment work. In contrast, criterion based tools compare the individual's performance with some significant standard and are concerned with the ability to perform certain specified and meaningful tasks. There are problems in applying normative tests to members of culturally different populations,(42,43) and these are accentuated when the total life experience of the individual has been quite unlike that of any single member of the normative group, as is true in the low birth weight infant.

Longitudinal studies in infancy(11) have drawn attention to two major discontinuities in normal psychological process, one corresponding to the expansion of retrieval memory at around eight months and the other to the acquisition of symbolic language just after the first birthday. These competencies (memory and language) change the nature of the psychological function as surely as, say, the transition from concrete to abstract thinking in pubescence. Moreover, the child who has been precocious in arriving at the first stage, when schemata for absent objects can be retrieved, is not necessarily going to be early in entering the second stage where language originates. Development must now be regarded as a journey in which one proceeds at different speeds over different parts of the route. I walk from my house to my car, drive to the airport, fly to Edmonton: running from my front door to the car will not make the plane arrive early. The implications for infant stimulation, both in terms of programming and evaluation, of this discontinuity, coupled with current re-interpretation of the importance of early experience,(44) are immense. Understanding of the transactional nature of development, of the effects upon parenting style of the early but transient apathy or hyper-arousal of the at-risk infant and the resulting change in social environment, forces a change in both assessment and intervention from a child-centred to an environment-centred, or at least equally balanced, approach.

What we should seek from tests, as we do from clinical neurology, is a capacity to describe the infant as he is now, to provide information which can be integrated with the whole array of multi-dimensional assessment data to answer immediate questions relating to his participation in developmental transactions.

Criterion-based measures, such as the Piagetian-cognitive techniques of Casatti and Lezine(45) or Uzgiris and Hunt,(46) should be judiciously intermixed with normative tools. Clinicians eagerly await field-applicable versions of some of Kagan's criterion measures in the independent and discontinuous areas of memory, self-awareness and the use of symbolization and deductive reasoning in linguistic growth.

Of the available normative measures, the Bayley scales(36) are now backed by unparalleled research experience, and are the yardstick against which the "test normality" of the individual can be judged. Such scales have limited sensitivity. Whilst the Bayley tends to be blind to social class effects, these have been demonstrated even in the sensori-motor period when such parameters as fixation time to discrepant or surprising events(47) are investigated. Wise psychometrists will not apply the motor portions of scales once locomotor deviance has been recognized - the concept of "motor age" has no place in the evaluation of the dystonic or cerebrally palsied child, and little meaning in any situation.

The question is often asked, "for how long should one 'correct' for prematurity by comparing the low birth weight infant with others of his post-conceptional (rather than chronological) age?" A very low birth weight infant four months after term cannot be compared with a normal four month old. Experience, maturation and environment have had little in common and this may be reflected in unique motor patterns and behaviour. Performance on normative tests "better than" corrected age cannot be expected. Normalization or "catch-up" most often occurs early in the second year of life, but may be delayed into the third.(48)

A further distinction is drawn between normative and ipsative testing. The latter considers variations over time in the qualities of a subject's performance of a standard scale. An accelerating progress rate is a more encouraging finding than steady slow gain, and the least one can expect of any intervention is that it should induce faster progress than maturation alone. Of course, without interventions designed to improve the transactional exchange, maturation may well seem to arrest. Demonstration of gains is important as a

SKILL ATTAINMENT SCALE
CRITERION TASK PERFORMED

Level 9 ...... Generalisation	spontaneously in play under any or varied conditions (mastery play)
Level 8 ...... Maintenance	reliably over sessions without specific programming
Level 7 ...... Competence	reliably (90%) within session without additional dues
Level 6 ...... Practice	reliably (50%) without additional cues
Level 5 ...... Verbal cueing	only with directions, "support" or reinforcement
Level 4 ...... Visual direction	only with modelling, gesturing and attentional redirection
Level 3 ...... Contact Prompting	only with tactile stimulation or other prompting short of full physical guidance
Level 2 ...... Physical Guidance	but requires moderate to maximal guidance in some input modality ± extrinsic reinforcement
Level 1 ...... Simplified	with simplified media or requirements
Level 0 ...... Unprepared	Developmentally unprepared to approximate task or sustain attention, or resists prompting

source of reward for parents and professionals alike, and may allow a reduction in program intensity.

The 'Skill Attainment Scale'(49) is an example of a graded qualitative rating which may be applied to any defined skill and used to monitor in-program advances or to determine when a given task approaches or achieves generalizability. Note that "training to test" is only useful if generalizability can be proven; note also that on normative tests, depending upon the rigour of the examiner and the total situation, an item might be passed at level four or five or even failed at level seven.

I wish all those involved in 'Infant Stim' were better grounded in basic psychometrics. Some developmental therapists - a term generally left undefined - act as though they did not know that half of all children will always be below average, or that one out of every six 18-month olds is "two months delayed". I wish that more respect were paid to the need for some parents to get on with their own job, and develop their own competencies in their own way, without professional help, especially after three months of intensive care, and that to do so is as likely to be a sign of coping adaptation as a warning of denial or disinterest.

I have said little of the medical contribution to assessment. It is essential. Generally health correlates more closely with infant development than do obstetric or neonatal events.(50) Medical insights ensure that both protective and high-risk factors are properly identified. The distinction between infants born too early and those born too small must be made. No efforts must be spared to eliminate medical conditions which are either treatable or progressive, or of which undesirable replication could be avoided by genetic action. Techniques of neurological examination are of less importance than that the findings can be interpreted from a developmental perspective and combined with multidisciplinary observations. Sophisticated analysis of movement patterns demands a combined neurodevelopmental and physical therapy approach. Useful procedures will focus on delay or deviance in evolution of tone and the pattern of primitive and postural reflexes and motoric integration. Assessment cannot be conducted without access to the full range of diagnostic investigations.

To sum up, assessment must be recognized as a shared information-gathering and decision-making process, involving parents and a team of professionals, dedicated to providing infants at risk with an optimal environment for learning, conscious of the full implications of the transactional exchanges between infant and caretakers, acknowledging a relationship to current developmental theory and operating within a framework of responsible accountability.

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THE ANATOMIC AND PHYSIOLOGIC BASIS OF NEUROLOGIC DEVELOPMENT IN THE PERINATAL PERIOD

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Neurologic development in the infant and child is fundamental to understanding paediatrics. Descriptive data are abundant, documenting the range of normal acquisition of motor skills, and extending into the neonatal period of prematurely born infants. The fundamental question we should no longer be asking is 'At what age should the normal child sit without support?', but rather 'Why can a child not sit before five or six months of age?'. With respect to the well-documented sequence of changes in muscle tone and posture in the preterm infant, the crucial questions also relate to the anatomic and physiologic mechanisms that explain the observed changes.

Development, after the initial period of neural tube formation and active neuronogenesis in the first weeks of embryonic life, depends upon three neuroanatomic processes: 1) neuronal migration, 2) synaptogenesis and 3) myelination. These processes continue well into the postnatal period, and, at the time of parturition, are not accompanied by any particular break or change in this progressive maturation. A fourth process, of a biochemical nature, is the biosynthesis of neurotransmitter substances.

Neuronal migration, from the subependymal germinal zone to the periphery of the cerebral cortex, begins at two months' gestation, and is not complete until five months postnatally in the term infant. In the cerebellar cortex, the external granular layer does not complete its migration into the interior of the folia until after the end of the first postnatal year. By contrast, most neuronal migrations within the brainstem are complete by the end of the first trimester of gestation. The formation of the convolutions of the cerebral and cerebellar cortices is directly related to neuronal migration.

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The development of each gyrus and sulcus is predictable and linked in time with gestational age. Because the cerebral cortex has a laminar organization, the formation of convolutions is an efficient method of increasing the surface area of the cortex without proportionately increasing the mass and volume of the brain.(1,2,3)

Synaptogenesis, the formation of synaptic relations between neurons to establish the circuits necessary for integrated cerebral function, is an on-going process that may completely end only in death in old age, and it is likely that some turnover of synapses occurs normally at all ages. In some parts of the brain, such as the cerebellar cortex, axons of external granular cells establish synapses with dendrites of Purkinje cells before migrating to their mature location. However, in the cerebral cortex, neuronal migration generally precedes synaptogenesis. The most active period of proliferation of dendritic spines and synaptogensis in the cerebral cortex is between 28 and 32 weeks gestation, a viable period for premature infants. The electroencephalogram undergoes important maturational changes during this period that probably reflect the formation of new synapses and intracortical circuits.(4,5,6) Synapses may be excitatory or inhibitory. This character is determined partly by the type of connection and partly by the type of chemical neurotransmitter involved. Axodendritic synapses in general are excitatory in nature; axosomatic and axoaxonic synapses generally are inhibitory.

Myelination of pathways in the central nervous system occurs in a predictable and time-linked sequence.(7,8,9) Some tracts begin myelination as early as 14 weeks gestation and complete their myelination cycles in a few weeks. Examples include the medial longitudinal fasciculus, spinal roots, and the auditory pathways, including not only the acoustic nerve, but also the trapezoid body, lateral lemniscus and brachium of the inferior colliculus. By contrast, the optic nerve does not begin to acquire myelin until near term. The large corpus callosum begins its myelination cycle at about four months postnatally, and does not complete it until mid-adolescence. Some association fibers connecting the frontal with temporal and parietal lobes of the same hemisphere are not completely myelinated until the fourth decade of life.

Myelin is not essential for conduction and some peripheral autonomic nerves remain unmyelinated throughout life, yet function normally. However, myelin is very important in most tracts of the central nervous systems for two reasons. First, it insulates individual nerve fibers to prevent short-circuits which may interfere with precision. Second, and of even greater importance, myelin greatly increases the speed of transmission by allowing salutary conduction between nodes of Ranvier in the propagation of action potentials along dendrites and axons, particularly in the long ascending and descending pathways. Slow conduction due to lack of myelination of tracts does not merely mean that young infants move and think more slowly than adults. It means that the possibilities of polysynaptic conduction in series is greatly reduced. As a hypothetic example, an unmyelinated nerve fiber may be capable of conducting a maximum of 50 impulses per second; with myelin sheathing, the same axon may be able to conduct 200 impulses per second. If the critical number of impulses per second needed for releasing a sufficient quanta of neurotransmitter at the synapse to depolarize the postsynaptic membrane of the next neuron in series is 100 impulses per second, it is apparent that many messages will be lost by synaptic block if single unmyelinated nerve fibers are involved. Convergence of two such unmyelinated fibers firing simultaneously will be able to produce an action potential in the next neuron in series, but this requires a more complex arrangement. The nervous system is organized on the basis of temporal summation of impulses to relay messages across synapses, and thus employs sequential series of neurons. Without myelin to increase the rate of conduction, the available circuits in the brain are severely restricted in number and lack the plasticity of alternative pathways. The complex human brain is reduced to a simple ganglion with the limited responses of a primitive worm.

The biosynthesis of neurotransmitters is probably the most important function of a neuron. Without this function, the neuron is isolated and unable to communicate with other nerve cells or participate in circuits that subserve complex functions. More than 26 distinct neurotransmitters have already been identified in the brain, and many other substances are suspected of being transmitters. Most transmitters are amino acids, some excitatory and others inhibitory, but each neuron produces a specific transmitter characteristic of the part of the brain to which it belongs. The depletion or impaired synthesis of these transmitters by neurons may be the reason for altered function in pathologic states such as following birth asphyxia.

It is instructive to examine pathologic conditions in the neonatal brain in relation to these normal developmental processes. In most cerebral malformations, abnormalities of neuronal migration is the primary pathogenesis, often induced by an environmental toxin or fetal infection, or due to faulty genetic coding as in chromosomal disease. In cretinism, the hypothyroid state leads to delayed myelination, although the sequence of myelination is not altered; malnutrition may lead to a similar delay in the processes of synaptogenesis and myelination. Birth axphyxia or ischemic-hypoxic encephalopathy causes an impairment of neuronal function and of the pathways arising from these neurons but, despite the diffuse nature of the insult, some parts of the brain are selectively more vulnerable than others due to differences in cerebral circulation and to metabolic differences. Thus hypoxic encephalopathy is a prototype of selective cerebral impairment from which we may better understand the function of the immature brain. Furthermore, ischemic-hypoxic encephalopathy is the most common neurologic disease in the perinatal period at all gestational ages.

The following discussion attempts to address a few important principles of neurologic development in the fetus and premature infant from the perspective of anatomy and physiology, and to relate them to alterations imposed by disease states, particularly the important clinical problem of encephalopathy due to birth asphyxia.

The sequence of neurologic development progresses at the same rate in the healthy infant, regardless of intrauterine or extrauterine environment.

The cardiopulmonary system undergoes profound anatomic and functional changes at the time of delivery. Less extreme changes occur in renal and gastrointestinal function, enzymatic induction in the liver, and the new role of the immunologic system after birth. The central nervous system, however, matures at the same rate, and delivery from the uterus does not change the rate or sequence of neuronal migration, synaptogenesis, or myelination. These anatomic substrates of cerebral maturation may be delayed or altered by disease which complicates prematurity, however, such as hypoxia, hypoglycemia, intraventricular or intracerebral haemorrhage, or hydrocephalus. While minor changes, such as the rate of secretion of cerebrospinal fluid, may be modified by delivery of the preterm or term fetus, the central nervous system including the special sensory pathways for vision, hearing, taste and olfaction, are not affected by the environmental change from the womb to the incubator. A six-week-old infant born at 34 weeks gestation should have the same neurologic examination as a term infant born yesterday.

Neurologic development is subject to individual biologic variation, which is greatest in neonates of advanced gestational age. Thus, just as some 'normal' children begin to walk at ten months of age and others do not walk until fourteen months, some neonates develop muscle tone and visual responses somewhat earlier than others. The neurologic examination is accurate to within a two week period in estimating gestational age, but individual biologic variation precludes more precise judgement.

The development of muscle tone in the premature infant begins in the lower extremities and proceeds in a cephalic direction.

This feature appears to be a paradox because the growth of the long axons of upper motor neurons proceeds rostrocaudally. Myelination of the largest descending motor pathway, the corticospinal tract, also proceeds rostrocaudally from the pons and midbrain cerebral peduncles, thus reaching the cervical part of the spinal cord sooner than it arrives at the lumbosacral region. This particular tract is atypical of motor pathways; others myelinate in a caudorostral direction.(2) Flexion of the legs precedes flexion of the arms in human development, however, giving the characteristic 'frog-like' posture of the 34-week premature infant. The sequence of maturation of individual types of axons within a tract, and the progression of maturation of spinal interneurons and their synaptic relations are incompletely understood, but probably provide an anatomic substrate for caudorostral development in the spinal cord just as in the brain. Neuronal organization and function develop sooner in the medulla oblongata than in the midbrain, and the most rostral centers of the forebrain last of all.

This aspect of neurologic development probably also has a phylogenetic origin because it is almost universal among vertebrates.(10) The hindlimb buds of the tadpole sprout and develop sooner than do the forelimb buds in the metamorphis of the frog. Tail movements are the earliest muscular activity of embryonic fishes. In a few mammals, such as the kangaroo, forelimbs develop earlier than hindlimbs and are used in the journey into the maternal pouch, but this example is probably a secondary evolutionary specialization in the divergent marsupials.(11)

Muscle tone develops sooner in extensor than in flexor muscles, but flexor tone ultimately predominates.

Postural changes in the preterm infant are a reflection of the development of muscle tone. They provide one of the most useful clinical criteria in the neurologic assessment of gestational age. The original observations of this predictable sequence of postural changes were made by the French paediatric neurologists, Andre-Thomas, Saint-Anne Dargassies, Amiel-Tison, Dreyfus-Brisac, Monod, and their associates.(12,13,14,15) Passive rather than active tone is more reliably assessed, because it is more independent of strength, and is determined by resting posture, angle of flexion, resistance to extension and passive recoil.(12) Similar observations were documented in the United States, England and Germany.(16,17,18,19) Scoring systems based on these descriptions were later devised by Dubowitz, et al., Ballard, et al., and others.(20,21,22) While these purely descriptive data retain clinical importance at the bedside, they may be expanded to understand the physiologic basis of such dynamic changes in tone and posture by examining the sequence of myelination in the developing human brain and correlating it with experimental studies in animals.

Many pathways originate from nuclei in the brainstem and descend in the spinal cord to modify the activity of motor neurons. Examples of such tracts include the rubrospinal, tectospinal, reticulospinal, vestibulospinal and oliviospinal. Furthermore, many of these descending pathways are anatomically further divided into two or more distinct bundles of nerve fibers, often travelling in medial and lateral parts of the brainstem. The large corticospinal tract originates in the cerebral cortex, and passes through the brainstem en route to the spinal cord, but is a much more complex pathway than its anatomic compactness suggests. In addition to mediating impulses arising from many parts of the cerebral cortex and in addition to the large contribution from the motor areas of the frontal lobe, the corticospinal tract is the final common pathway over which the cerebellum and corpus striatum influence muscle tone and coordination. Direct cerebellospinal and striatospinal pathways do not exist in man or other mammals, and the efferent projections from the cerebellum and from the globus pallidus (which relays impulses from the candate nucleus and putamen) ascend to the thalamus and then to the cerebral cortex to be integrated and carried to the spinal motor neurons via the corticospinal tract.

Lawrence and Kuypers carried out a series of experiments in newborn monkeys, selectively stimulating or destroying small zones of the brainstem through which the various descending pathways pass.(23,24) They concluded from their observations of the effects on muscle tone and posture of these animals that all descending motor tracts could be physiologically grouped into three pathways in terms of their effects on selective motor neuron activity. They termed these groups the medial subcorticospinal, lateral subcorticospinal, and corticospinal pathways. The medial and lateral subcorticospinal pathways were named for their positions in the brainstem, near the midline or near the periphery, respectively, and each incorporated several anatomically distinct tracts such as the rubrospinal and vestibulospinal. The net physiologic effects of each of these grouped pathways may be summarized as:

The medial subcorticospinal pathway causes contraction of proximal extensor muscles and contraction of distal flexor muscles.

The lateral subcorticospinal pathway has an inhibitory effect on the same phasic alpha motor neurons strongly influenced by the medial subcorticospinal pathway.

The corticospinal tract has effects antagonistic to those of the medial subcorticospinal pathway; it causes proximal flexion and distal extension.

Examination of the sequence of myelination in the premature infant reveals an explanation of the changes of posture with advancing conceptional age, if the observations of Lawrence and Kuypers are considered. The medial pathways myelinate sooner than do the lateral tracts of the brainstem, and the corticospinal tract myelinates latest of all. The infant of 30 weeks gestation thus lies fully extended, because only the medial subcorticospinal pathway is partially myelinated. The hips may be abducted rather than extended because of the physical structure of the immature hip joint, but abduction and extension are neurologically paired, and adduction and flexion are neurologically similar. By 34 weeks gestation, the tracts composing the medial subcorticospinal pathway are fully myelinated, and those of the lateral subcorticospinal pathway are now partially myelinated.(8,9) Myelination proceeds in the corticospinal tract, although it is not complete in this pathway until two years of age. By term, 40 weeks gestation, both subcorticospinal pathways are fully myelinated and, in the corticospinal tract, myelin is well demonstrated at the level of the cerebral peduncles of the midbrain and in the upper pons, but not by light microscopy at the level of the medulla. Electron microscopy reveals two or three turns of myelin around each axon in the pyramids of the medulla, however.(2)

These observations offer insight into the postures of infants with brain damage. In anoxic encephalopathy due to birth asphyxia, one of the most constant features is strong adduction of the thumb and flexion of the fingers and toes.(25) In severe cases, extension or abduction of the hips and extension of the spine or 'opisthotonus' may occur. These postures are related to the greater vulnerability of the cerebral cortex than the brainstem to functional impairment associated with ischemic or hypoxic insults. The medial subcorticospinal pathway continues to exert a strong influence, but is not antagonized or balanced by activity of the corticospinal tract. The clinical features reflect this loss of equilibrium in the system. The inhibitory effects of the lateral subcorticospinal pathway are not strong enough to completely balance the medial subcorticospinal pathway alone.

In normal neurologic development, progressive flexion occurs in proximal joints, and extension of fingers and toes is better developed in infants at term. The intermediate joints, such as elbows and knees, are neurologically more closely associated with proximal joints such as shoulders and hips, because most of the muscles that move them are located in upper segments of the extremities, but, from the neurologic standpoint, the postures of the elbows and knees are of less importance in assessing neurologic maturation than are the fingers, toes, shoulders, hips and spine.

The full-term newborn infant may be thought of as in a physiologic state of 'spasticity'. Although it may be inappropriate to employ a term denoting a pathologic state to describe a transitional physiologic condition, it is instructive for purposes of understanding the incomplete myelination of the corticospinal tract at birth to consider the similar characteristics of the normal neonate with the abnormal patient of later infancy or childhood who has spastic diplegia or 'cerebral palsy'. Both have increased passive muscle tone relative to the normal tone of late infancy and childhood. Both have overly brisk tendon stretch reflexes, cross adductor reflexes, a few beats of ankle clonus and extensor plantar responses. The neonate even has transitory 'flexion contractures' with a more limited range of motion of elbows, knees and hips than he will have later. Fine finger coordination is impaired in the spastic child and is less precise in the normal neonate than it will be in later infancy, although individual finger movements seen at birth will disappear for a few months to return in more precise form for other reasons explained later in this treatise. The corticospinal tract thus is progressively more functional after 32 weeks gestation and is active in the term neonate, but is not yet fully mature and results in many clinical features resembling those of the older child with corticospinal tract deficit or damage to the cerebral cortex from which this important pathway arises.

It is important to recognize that the corticospinal tract is really an inhibitory pathway. Inhibition is an active, not a passive, phenomenon in the central nervous system. When asked how such an important motor tract could have a negative effect, Sir John Eccles, the Australian neurophysiologist who discovered inhibitory pathways, is said to have replied that the nervous system is like a sculpture rather than a painting. Just as Michaelangelo created the statue of David from a massive block of stone by selectively chipping away from, not adding to, that stone, so the corticospinal tract creates refined movement from massive excitation by selective inhibition of certain combinations of motor neurons. Uninhibited diffuse excitation is spasticity, a clinical expression of corticospinal tract deficit in the older child.

Other factors influencing muscle tone and posture include vestibular reflexes and the activity of the corpus striatum and cerebellum.

The 'asymmetric tonic neck reflex' is called 'asymmetric' not because the extremities on the side to which the head is turned extend while the arm and leg of the opposite side flex, but because it is different in nature on the right and left. It is a vestibular reflex mediated in part by the labyrinths and in part by stretch receptors in cervical muscles. This reflex appears at about 32 weeks gestation and is so strong and reproducible by 36 or 37 weeks that it appears mechanical, as if the infant had gears in his neck. Thereafter it diminishes in strength and habituates, so that the full-term neonate may assume an incomplete tonic neck posture when the head is briskly turned by the examiner, but it is not reproducible on successive turning of the head. A strong, non-habituating asymmetric tonic neck response in a term newborn is a pathologic reflex. The position of the head should be noted in examining tone and posture in the infant, particularly if an asymmetry is found, and the head should be turned if any difference between right and left sides is found, before hemiparesis is considered.

The corpus striatum, consisting of the putamen and caudate nucleus, and the global pallidus influence muscle tone and posture, but only recently have been recognized to play an important role in the newborn. The investigations of the late Professor Derek Denny-Brown demonstrated that small bilateral lesions in the posterior part of the putamen of neonatal monkeys resulted in strong active flexion of all the extremities, both proximally and distally, and that this posture could not be overcome by vestibular reflexes or tactile stimulation.(11) The corpus striatum mediates its effects through the corticospinal tract. The main efferent pathways from the globus pallidus to the thalamus, ansa lenticularis and ansa fascicularis, are well myelinated at term. The effects of the corpus striatum on tone and posture probably are important only late in gestation.

The cerebellum is another structure of incompletely understood importance in the maintenance of neonatal muscle tone and posture. Infants with cerebellar hypoplasia are profoundly hypotonic from birth,(26) and this finding is due to decreased fusimotor activity of muscle spindles.(27)

Motor pathways other than the corticospinal tract are of relatively greater importance in the neonate than at any other age.

The corticospinal tract is the largest and most important of the descending motor pathways in man, without which severe spastic diplegia results. Voluntary control of refined movements, especially of the fingers, is lost in patients with lesions of the motor cortex or corticospinal (pyramidal) tract. In monkeys, sectioning the pyramidal tract results in impairment or loss of individual finger movements, those of extension being more severely affected than those of flexion; pincer grasp between the thumb and index fingers is profoundly impaired.(28) These observations confirm the earlier observations of Lawrence and Kuypers.(23,24) However, if less than 80% destruction of the pyramidal tract occurs, the monkeys may achieve recovery of function of the fingers in time.(29) Since nerve fibers do not regenerate in the central nervous sytem, alternative pathways must become established to subserve these same functions.

The corticospinal tract is an exclusively mammalian structure without a counterpart in non-mammalian vertebrates.(10) Despite the absence of this pathway, fishes, amphibians, reptiles and birds all are capable of rapid, accurate and precise motor activity. Consider the precision needed by a frog in snatching a fly in midair, of a rattlesnake in its lethal strike, or of a bird alighting on a narrow branch. These refined skills are accomplished by motor pathways arising from structures of the brainstem. In the frog, for example, the optic tectum is a highly differentiated structure that subserves not only vision, but also is a centre of correlation for sensory information from the somatosensory and vestibular systems and translates these data into appropriate behavioral responses. The optic tectum of the frog thus performs many functions of the cerebral cortex in mammals including the initiation of motor responses, and the tectospinal tract substitutes for the corticospinal tract of man in that species. The red nucleus and rubrospinal tract are other important structures conveying motor impulses in non-mammalian vertebrates.(10)

These evolutionary aspects of the phylogenetic development of the brain provide an explanation of some features of human neurologic development in infancy. Individual movements of fingers are a characteristic of the term neonate, but are later lost as 'mitten grasp' develops at three to four months of age, the four fingers acting together as a unit. Only later, at eight months, are individual finger movements reacquired, but now with much greater refinement and voluntary control than the young infant possesses. The corticospinal tract is certainly functional in the neonate in influencing muscle tone and posture, but is not yet well enough myelinated at birth to provide for much voluntary control of movements. As in lower vertebrates, the motor structures of the brainstem such as the red nucleus, optic tectum (superior colliculus) and vestibular nuclei have a relatively greater importance in the neonate than they will have at any other time in life. The 'subcorticospinal' pathways provide the refinement of motor activity, including movements of the eyes, tongue, palate and extremities that will later be superceded by the corticospinal tract. As myelination of the corticospinal tract proceeds during in the first year, increasing function of the pathways first suppresses the functional expression of the subcorticospinal pathways, and then replaces it with similar function carrying much greater ultimate potential for refinement of motor behaviour and voluntary control of movement. The young infant thus transiently appears to regress in individual finger movements as corticospinal control replaces brainstem control. Some reflexive motor activity is never completely inhibited by the domination of the corticospinal tract, but this pathway and the cortical motor centers of its origin allow a flexibility of volitional motor control not possible using brainstem centres alone.

Our understanding of motor control in early infancy is incomplete and rudimentary, but at least provides a beginning of scientific explanation. We must be cautious to guard against invoking the 'amazing newborn' notion. This fantasy embodies the newborn infant with magical qualities of intellect and powers of perception and motor control beyond reality, which are then mysteriously lost as the infant matures. The net effect of such mystique is to cloud accurate observation and retard scientific explanation of reproducible clinical findings.

Stretch reflexes are normally inhibited and tactile reflexes are reinforced by cerebral cortical influence in the newborn.

The corticospinal tract, as an inhibitory pathway, has a direct effect on spinal motor neurons by diminishing the strength of the simple monosynaptic reflexive loop between skeletal muscle and spinal cord. Infants with cerebral cortical impairment thus have uninhibited, overactive knee jerks, ankle clonus and generalized increased stretch reflexes.

Tactile reflexes are motor responses elicited by cutaneous tactile stimulation rather than stretch of a muscle or proprioception. Examples include the grasp reflex, rooting response, and trunk incurvation reflex of Galant, but the most important tactile reflexes the newborn infant possesses are the sucking and swallowing reflex. Although these reflexes are primarily neural pathways through the brainstem and spinal cord, the influence of the cerebral cortex upon them is the opposite of that on the stretch reflexes; it reinforces or strengthens the tactile reflexes.

Infants with cortical impairment or damage at birth regardless of cause, whether from meningitis, hydrocephalus, asphyxia, trauma, or congenital infection, all have one feature in common; they all are problem feeders. They suck poorly or even require gavage. Their coordination of the swallowing mechanism is impaired and they frequently aspirate milk and pool their own secretions in their oropharynx. After a few days or weeks, depending on the severity of the insult, these functions improve as their cerebral cortex recovers. Occasionally an infant with a severe cerebral malformation or other extensive cortical defect since early gestation exhibits a strong suck and feeds well. If the brainstem mechanisms mediating sucking and swallowing have not been modified by cortical influence in fetal life, these tactile reflexes may not be impaired. Those infants who abruptly lose cerebral cortical function after having possessed it in fetal life, as with birth asphyxia or neonatal meningitis, are the most vulnerable to manifest weak or absent tactile reflexes.

The grasp reflex is a tactile reflex, but often it is confused with the traction response, which is a tendon stretch reflex augmented rather than diminished by mild cortical insults. The classic photograph of a supine neonate grasping the fingers of an examiner and thus pulling himself to a sitting position is a dramatic example of this traction response; it is not a grasp reflex as it is often purported to be.(16) An alternative test of a tactile reflex in the hands is gently stroking the dorsal surface of the hand and fingers; this stimulus normally produces reflexive extension of the fingers in the newborn.

Tactile reflexes of the brainstem continue to require reinforcement from the cerebral cortex even in adults. The latency of the human corneal reflex is prolonged and the reflex weakened by cerebral cortical lesions such as infarcts.(30) The lower post-central gyrus has an excitatory influence on interneurons of the laternal reticular formation of the lower brainstem mediating trigemino-facial connections of the corneal reflex. These descending fibers from the cortex are conveyed via the cerebral peduncle as with the corticospinal tract.

An intact gamma loop is essential for the maintenance of muscle tone at all ages. Neuropathy or myopathy conceal or abolish the clinical expression of cerebral maturation on muscle tone and posture.

Spinal motor nerve roots are among the earliest axons to acquire myelin, beginning at 24 weeks gestation by light microscopic observation.(9) Their short myelination cycle is complete within two or three weeks. Neuromuscular junctions begin to form as early as the ninth week of embryonic life in man. Striated muscle is histologically and histochemically mature by 28 weeks gestation.(31) The early maturation of the neuromuscular system signifies that the changes observed in muscle tone and posture after 28 weeks gestation cannot be directly attributed to immature peripheral motor units, and must be central in origin.

The gamma loop is the feedback loop between spinal cord and skeletal muscle. An interruption or impairment of these afferent and efferent connections results in flaccid muscles and loss of tendon stretch reflexes. Diseases of motor neurons or of peripheral nerves beginning in utero, such as Werdnig-Hoffmann disease, do not allow the progressive changes in tone and posture to be expressed, even though the central nervous systems pathways myelinate at the normal rate. Primary myopathy at birth, such as neonatal myotonic dystrophy, or congenital neuromuscular diseases such as congenital muscle fiber type disproportion or nemaline rod disease also may conceal the normal sequence of motor maturation.

Segmental myoclonus ('jitteriness') may result from depletion or impaired biosynthesis of serotonin in the brain, and is not simply clonus.

A clinical phenomenon commonly observed in neonates who have suffered birth asphyxia or other cerebral insults is known to the paediatrician as 'jitteriness' or 'tremors', and to the neurologist as 'segmental myoclonus'. It consists of repetitive rhythmic contractions of extremities. Because of its shifting multifocal character, it is sometimes confused with seizure activity, although the electroencephalogram shows no paroxysmal activity synchronous with it and, unlike true seizures, it can be stopped by simple physical restraint. It is not modified by anticonvulsant drugs.

The pathogenesis of this condition may be attributed to two causes. The first is simple clonus, or an uninhibited stretch reflex in which each contraction is the stretch stimulus for the next, and the inhibitory influence of the corticospinal tract is lacking and therefore does not dampen the resulting oscillations. The phenomenon may be initiated by eliciting a knee or ankle jerk, or may start spontaneously with the infant's own sudden movements which produce a stretch reflex in his muscles.

Segmental myoclonus at times may also be observed in infants who do not have overactive tendon stretch reflexes, suggesting that the phenomenon involves additional factors than simply overactive stretch reflexes or clonus. Depletion of the central neurotransmitter 5-hydroxytryptamine, or serotonin, results in segmental myoclonus in experimental animals, especially immature or young subjects.(32) The myoclonus is ameliorated by administration of the serotonin precursor L-5-hydroxytryptophan. Some human patients with segmental myoclonus have been demonstrated to have low concentrations in cerebrospinal fluid of 5-hydroxindoleacetic acid, the primary metabolite of serotonin, suggesting a deficiency. Further support of this hypothesis is provided by evidence that serotonin antagonists block the development of L-5-hydroxtryptophan-induced myoclonus.(32) In the human brain, serotonin is the principal transmitter in the midline raphe nuclei of the brainstem, and also in parts of the thalamus.

Segmental myoclonus does not require treatment, usually resolves spontaneously after a few hours or days, and is not dangerous for the infant. It must be distinguished from seizures, however, which may or may not also occur in the infant. If the depletion of the neurotransmitter serotonin following hypoxia is important in its pathogenesis, however, it provides a prototype of a pharmacologically treatable specific disorder in postanoxic encephalopathy. Perhaps other more important depletions or impaired biosynthesis of neurotransmitters due to anoxia will be amenable to specific replacement therapy, analogous to the use of L-DOPA in Parkinson's disease.

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RETROLENTAL FIBROPLASIA - A HANDICAPPING CONDITION OF PERINATAL ORIGIN

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Since the identification of excessive oxygen administration as a cause in the production of retrolental fibroplasia (RLF) in the premature infant, it is the impression of the majority of the medical profession that retrolental fibroplasia is an extinct disease. In fact, it is not extinct but is of increasing incidence in civilized jurisdictions that save premature infants. It has been estimated that in the year 1979 in the United States from 400 to 800 children were blinded by retrolental fibroplasia and 2,000 or more experienced visual disability.(1) The prime reason for the increasing incidence is the increased salvage rate of the very low birth weight prematures. This is in spite of the better control of oxygen administration.

Of 100 prematures of 1,500 grams birth weight or less, approximately 70% will develop acute retrolental fibroplasia or retinopathy of prematurity (ROP). Of 100 prematures with ROP, in 85 the ROP will be mild and spontaneously regress with little or no visual disability. The remaining 15 with severe progressive ROP will develop significant degrees of cicatricial RLF. Three will be blind in both eyes, and the remaining 12 will be visually disabled with problems ranging from simply requiring glasses to having visual impairment, even with glasses, requiring special educational facilities and special visual aids. In addition to high refractive errors and anisometropia, there will also be a high incidence of strabismus, and later, retinal problems such as detachment in this group.

The only jurisdiction reporting a decreased incidence of blindness that is due to RLF is Japan.(2) Since 1977, on the report of the Joint Committee for the Study of RLF in Japan, they have a national classification of the disease in its acute and chronic phases and have recommended prophylactic treatment of severe progressive ROP with photocoagulation or cryotherapy.(3)

Clinical experimentation with prophylactic treatment has taken place outside Japan. The treatment, although destructive to peripheral retina and compromising some visual field, would certainly be indicated for the preservation of the majority of the field and intact central visual acuity. Some evidence suggests treatment is possible and beneficial,(4-10) other denies any beneficial effects,(11) and some suggests the possibility of detrimental effect.(12) The critical problem is the identification of which case should be treated and the timing of the treatment. Whereas 12 of the 15 with severe ROP will regress spontaneously, leaving mild to moderate cicatricial RLF, and may do as well as the treated cases of ROP, and whereas the accurate identification of the three of the 15 who will progress to blindness is a very difficult clinical estimation during the acute or ROP stage, accordingly the treatment is considered experimental in most areas outside of Japan.

Encouraging evidence has been presented of the efficacy of Vitamin E in the reduction of severity of ROP and accordingly of RLF, although it does not seem to affect the incidence of ROP.(13,14,15) Severe RLF is not eliminated, however, and the potential of surgical intervention must continue to be explored.

The answer to these questions will possibly be derived from a multi-institutional study comparing the results of large populations of non-treated cases of ROP to populations where treatment is employed. A fundamental prerequisite to such a study is the need for a common language, a common classification of RLF (ROP) to enable critical analysis of the natural and altered course of the disease.

In conclusion, retrolental fibroplasia is not an extinct disease. It is an increasingly significant and potentially preventable handicapping condition of perinatal origin.

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THE DENVER APPROACH TO DEVELOPMENTAL SCREENING AND DIAGNOSIS OF INFANTS AND PRE-SCHOOL CHILDREN

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One of the major problems plaguing educators and health professionals is the relatively high prevalence of school learning problems, many of which could have been identified during the formative infancy and pre-school years. Such problems are of major concern since they affect an average of 20 to 25% of the school-aged population and 40-45% of children residing in poverty circumstances (also referred to as low socio-economic status). The situation is particularly alarming since most of the learning problems could be avoided or minimized through earlier detection and remediation.

Our approach to this problem began in 1964, and was aimed at accurate and early identification of "high risk" children through the application of screening procedures to all children regardless of socio-economic status (SES). With the recognition that developmental problems may arise at any time during the first few years of life, our screening procedures were designed for repeated application from birth to six years of age.

Screening basically involves the use of quick and easily administered tests on an asymptomatic population to differentiate those who probably have the condition being screened for from those who do not. Screening tests are not diagnostic; a child identified as suspect by a screening test requires a diagnostic evaluation to determine whether or not he or she has the condition in question.

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In 1967 we came out with our first screening test, the Denver Developmental Screening Test or DDST (Frankenburg & Dodds, 1967). The test was standardized on 1,036 children ranging in age from two weeks to six years old. The screening test consists of 105 items arranged in four categories of functioning: personal-social, fine motor-adaptive, language and gross motor. Each item is represented by a horizontal bar placed along an age continuum. For example, Figure 1 illustrates the item "walks well".

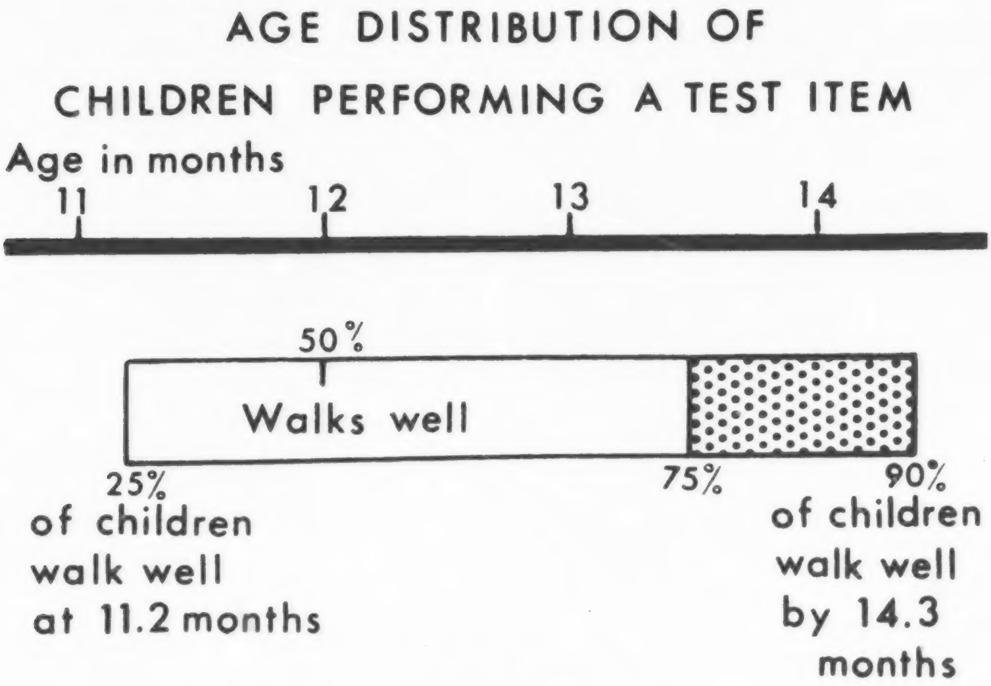

AGE DISTRIBUTION OF
CHILDREN PERFORMING A TEST ITEM
Age in months
11
12
13
14
50%
Walks well
25%
of children
walk well
at 11.2 months
75%
90%
of children
walk well
by 14.3
months

Figure 1

The left end of the bar indicates the age (11.2 months) at which 25% of the children can walk well; the hatch mark at the top of the bar indicates the age (12 months) at which 50% of the children can walk well; the left end of the shaded area indicates the age (13.5 months) at which 75% of the children can walk well; and the right end of the bar indicates the age (14.3 months) at which 90% of the children can walk well. Each of the 105 items is arranged within one of the four sectors on the test form (Figure 2).

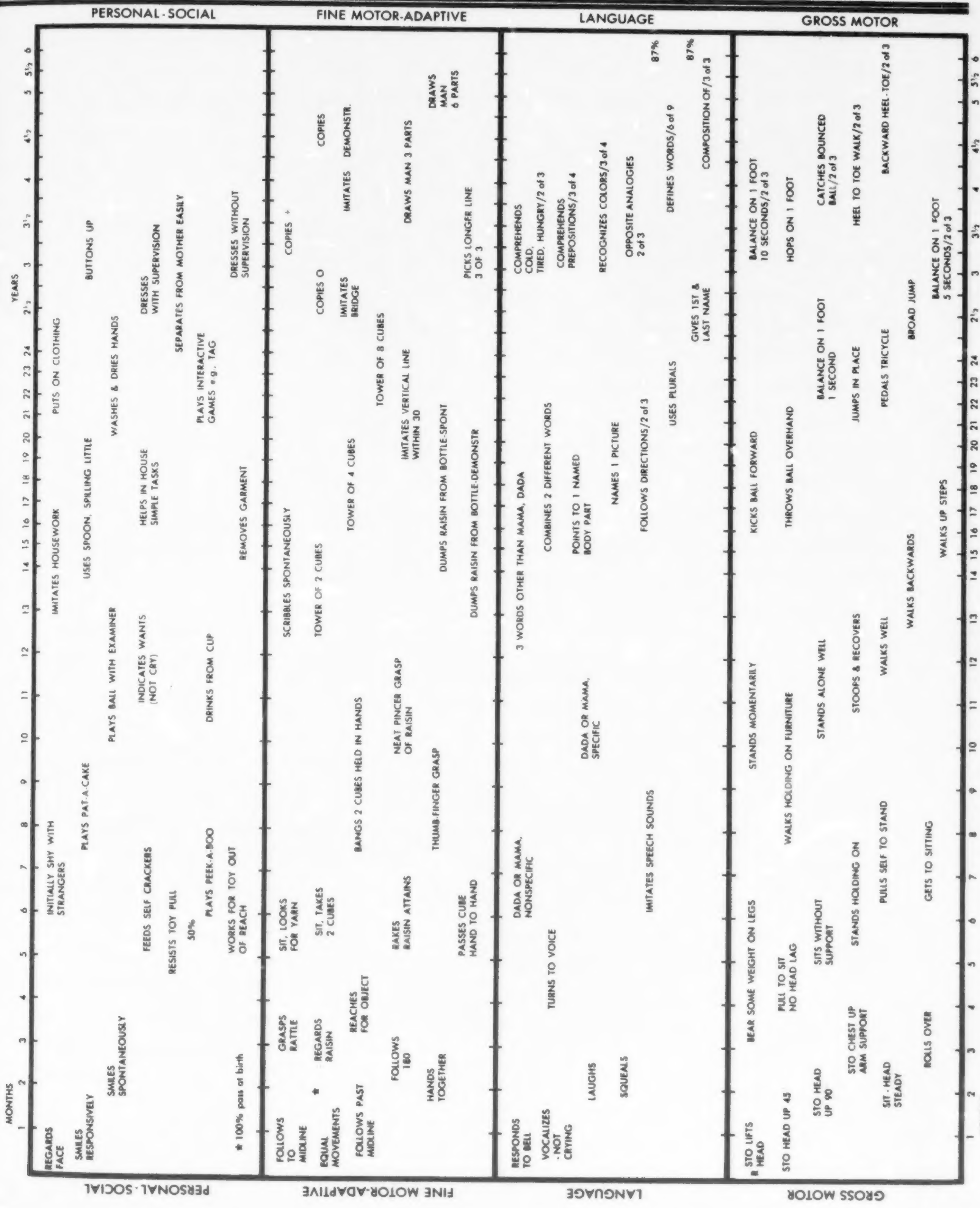
DENVER DEVELOPMENTAL SCREENING TEST

STO.=STOMACH
SIT=SITTING

PERCENT OF CHILDREN PASSING
May pass by report
25 50 75 90
Test Item
Footnote No. - see back of form

Date
Name
Birthdate
Hosp. No.

Figure 2



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Approximately 20 age-appropriate items are administered to determine if a child passes the majority of tasks passed by 90% of children in the normative sample. Using the scoring criteria described in the reference manual, the child's performance is classified as "normal", "abnormal", "questionable" (or borderline), or "untestable". All children who receive results other than normal should be rescreened one week later to rule out temporary effects, such as illness, hunger, fatigue or fear caused by separation from the parent.

Studies over the past 10 years have demonstrated that the DDST meets most of the criteria of the ideal screening test: reliability, validity, appropriateness for the population being screened and simplicity of administration. For instance, tester-observer and test-retest reliability were 97.7% and 91% respectively.

In regard to validity, health aides screened more than 2,000 children using the DDST. Two hundred and forty-six of the children also received either the Revised Bayley Infant Scale or the Stanford-Binet (see Table 1). It was determined that the aides had correctly identified 92% of the children with intelligence and developmental quotients below 70 (that is to say, in the retarded range).

Concurrent Validity of DDST

DDST (Revised Method of scoring)	D.Q./I.Q. Revised Bayley or Stanford-Binet			
	Abnormal below 70	Borderline 70 to 90	80 and above	Total
Abnormal	12	2	6	20
Questionable	1	17	36	54
Normal	0	10	162	172
TOTAL	13	29	204	246

Table 1

In regard to predictive validity, 89% of children receiving abnormal DDST results and 63% of children receiving questionable results failed in school five to six years later (see Table 2).

Results of DDST School Follow-up Study

DDST vs. Follow-up

DDST	Follow-up Status		
Revised Method of Scoring	School Problems	Normal	Total
Abnormal	31 (89%)	4 (11%)	35
Questionable	41 (63%)	24 (37%)	65
Normal	19 (38%)	32 (62%)	51
TOTAL	91	60	151

Table 2

A study involving more than 2,000 children demonstrated substantial differences in development for Anglo children whose parents were unskilled workers versus a cross section of Anglo children. Few differences between Anglo, Black, and Chicano children of the same socio-economic status families were shown. Thus, the test is not racially biased.

While the DDST is simple to administer, it is important to emphasize the necessity of proper training and monitoring which, after all, are necessary for most screening endeavors. To facilitate such training at minimum cost, a series of instructional training programs for physicians, nurses and lay aides have been developed and found to be highly effective.

It would appear from the foregoing that the DDST meets the criteria of the ideal screening test in view of its reliability, validity, appropriateness to most populations and ease of administration. The chief drawback to its use to screen large masses of children at periodic intervals is the 15-20 minutes required to screen each child. This problem has been overcome through use of a two-stage screening process depicted by Figure 3. In such a two-stage process, the first stage is quick and simple, making it economical; it must also be valid or accurate in identifying all of the developmentally-handicapped children screened. For such a process to work, it is essential that

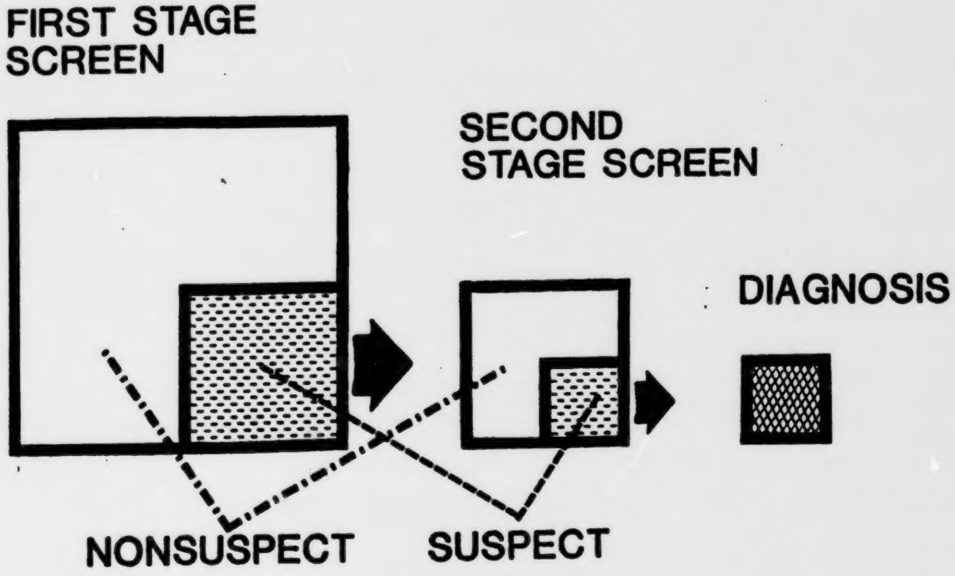


Figure 3

if errors occur in the first stage, they should be errors of over-referral rather than under-referrals. This is important, since under-referrals would not be identified and over-referrals would be decreased with the second stage screen. In such a two-stage process, all of those children who are suspect on the first stage screen are subject to the more lengthy, complex and therefore costly second stage screen.

Our recommendation for two-stage developmental screening consists of an abbreviated DDST for which one only needs to administer 12 age-appropriate items rather than the usual 20-25 items (see Figure 4). If the child fails to pass all 12 items, one administers the remaining 10 or so items to complete the DDST. The first stage, consisting of the 12 items is highly sensitive, in that it identifies all of the suspect children in about half the usual time required to administer the full DDST. In random samples, only about 25% of children were shown to be suspect on the first stage, and thus required the completion of the full DDST.

To simplify the administration of the DDST, the DDST form has been revised. In the revised form, all of the items which are contained in one of four sectors are arranged in a stepwise manner according to the age at which 90%

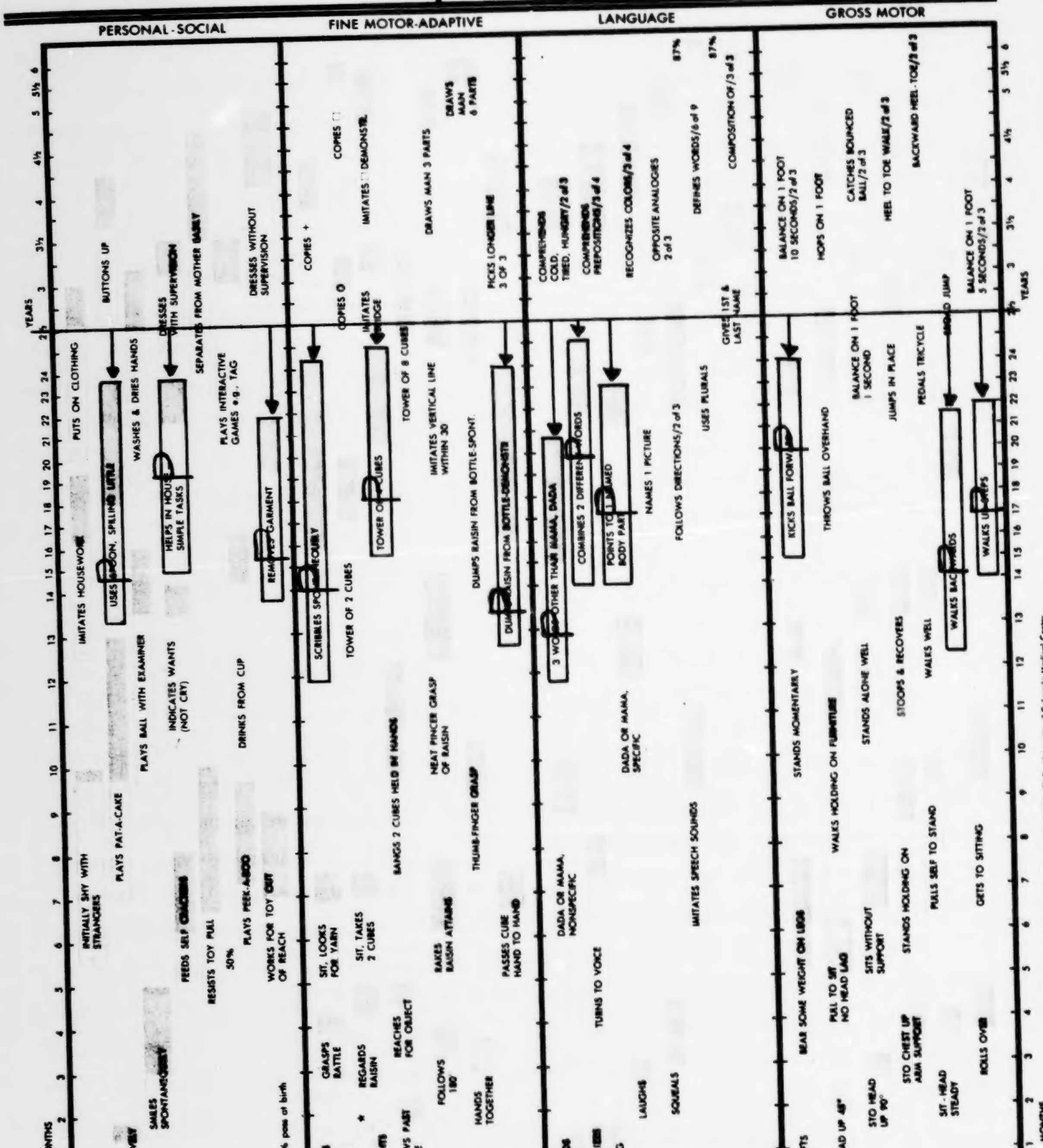
Figure 4

DENVER DEVELOPMENTAL SCREENING TEST

STO.=STOMACH
SIT=SITTING

PERCENT OF CHILDREN PASSING
25 50 75 90
May pass by report
Footnote No. see back of form
Test Item

Date
Name
Birthdate
Hosp. No.



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of children passed each item. Figure 5 depicts the revised test form, which is similar to a growth curve and has the advantage of depicting a child's developmental progress over time.

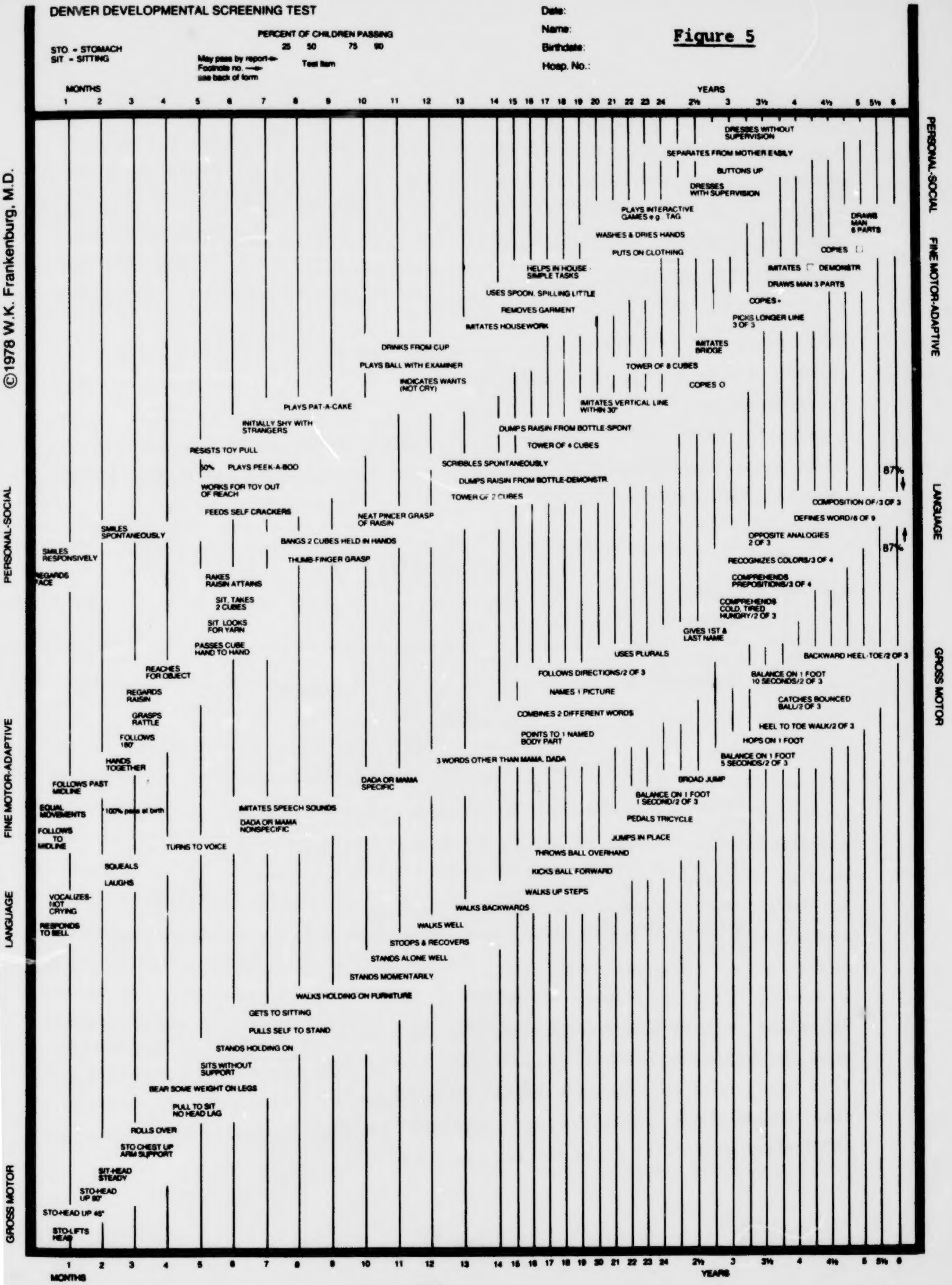
Since the home environment of children is a major predictor of later development (in fact it has been demonstrated to modify such developmental predictors as prematurity, hypoxia and others), it is important to also consider the environmental status of children being screened. Such a consideration is particularly important, if one is interested in predicting later school failure among low SES children where the prevalence of school failure may be as high as 43%. This percentage is markedly higher than that for the more privileged children residing in high SES families where school failure is generally less than 18%. After much deliberation and in recognition of the need to be selective in screening, we in Denver have decided to screen children to identify those who are at high risk for later school failure.

The reasons for targeting on school failure as the purpose of our screening efforts are as follows:

1. School failure can be prevented.
2. Early diagnosis and treatment improve outcome.
3. Early identification and treatment are cost effective.
4. School failure is a highly prevalent problem.

While the DDST reflects the child's current developmental status, as it is dependent upon the child's biological integrity and experience, the environmental evaluation - particularly for low SES children - may increase predictive accuracy by determining if the home environment is likely to compensate for developmental deficits or further delay future development. Thus, a child who is abnormal on the DDST and who comes from a home that does not foster development is at double risk for school failure, while a child receiving a normal DDST evaluation and who comes from a home that is promoting development is at minimal risk for later school failure. (See Figure 5.)

Figure 5


DENVER DEVELOPMENTAL SCREENING TEST
Date:
Name:
Birthdate:
Hosp. No.:
STO. = STOMACH
SIT. = SITTING
PERCENT OF CHILDREN PASSING
25 50 75 90
May pass by report
Footnote no. see back of form
Test Item
MONTHS
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
YEARS
2½ 3 3½ 4 4½ 5 5½ 6
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PERSONAL-SOCIAL
FINE MOTOR-ADAPTIVE
LANGUAGE
GROSS MOTOR
DRESSES WITHOUT SUPERVISION
SEPARATES FROM MOTHER EASILY
BUTTONS UP
DRESSES WITH SUPERVISION
PLAYS INTERACTIVE GAMES e.g. TAG
WASHES & DRIES HANDS
PUTS ON CLOTHING
HELPS IN HOUSE - SIMPLE TASKS
USES SPOON, SPILLING LITTLE
REMOVES GARMENT
IMITATES HOUSEWORK
DRINKS FROM CUP
PLAYS BALL WITH EXAMINER
INDICATES WANTS (NOT CRY)
PLAYS PAT-A-CAKE
INITIALLY SHY WITH STRANGERS
RESISTS TOY PULL
50% PLAYS PEEK-A-BOO
WORKS FOR TOY OUT OF REACH
FEEDS SELF CRACKERS
SMILES SPONTANEOUSLY
SMILES RESPONSIVELY
REGARDS FACE
DRAWS MAN 6 PARTS
COPIES □
IMITATES □ DEMONSTR.
DRAWS MAN 3 PARTS
COPIES +
PICKS LONGER LINE 3 OF 3
IMITATES BRIDGE
TOWER OF 8 CUBES
COPIES O
IMITATES VERTICAL LINE WITHIN 30°
DUMPS RAISIN FROM BOTTLE-SPONT.
TOWER OF 4 CUBES
SCRIBBLES SPONTANEOUSLY
DUMPS RAISIN FROM BOTTLE-DEMONSTR.
TOWER OF 2 CUBES
NEAT PINCER GRASP OF RAISIN
BANGS 2 CUBES HELD IN HANDS
THUMB-FINGER GRASP
RAKES RAISIN ATTAINS
SIT, TAKES 2 CUBES
SIT, LOOKS FOR YARN
PASSES CUBE HAND TO HAND
REACHES FOR OBJECT
REGARDS RAISIN
GRASPS RATTLE
FOLLOWS 180°
HANDS TOGETHER
FOLLOWS PAST MIDLINE
EQUAL MOVEMENTS
100% pass at birth
FOLLOWS TO MIDLINE
87%
COMPOSITION OF/3 OF 3
DEFINES WORD/6 OF 9
OPPOSITE ANALOGIES 2 OF 3
87%
RECOGNIZES COLORS/3 OF 4
COMPREHENDS PREPOSITIONS/3 OF 4
COMPREHENDS COLD, TIRED HUNGRY/2 OF 3
GIVES 1ST & LAST NAME
USES PLURALS
FOLLOWS DIRECTIONS/2 OF 3
NAMES 1 PICTURE
COMBINES 2 DIFFERENT WORDS
POINTS TO 1 NAMED BODY PART
3 WORDS OTHER THAN MAMA, DADA
DADA OR MAMA SPECIFIC
IMITATES SPEECH SOUNDS
DADA OR MAMA NONSPECIFIC
TURNS TO VOICE
SQUEALS
LAUGHS
VOCALIZES-NOT CRYING
RESPONDS TO BELL
BACKWARD HEEL-TOE/2 OF 3
BALANCE ON 1 FOOT 10 SECONDS/2 OF 3
CATCHES BOUNCED BALL/2 OF 3
HEEL TO TOE WALK/2 OF 3
HOPS ON 1 FOOT
BALANCE ON 1 FOOT 5 SECONDS/2 OF 3
BROAD JUMP
BALANCE ON 1 FOOT 1 SECOND/2 OF 3
PEDALS TRICYCLE
JUMPS IN PLACE
THROWS BALL OVERHAND
KICKS BALL FORWARD
WALKS UP STEPS
WALKS BACKWARDS
WALKS WELL
STOOPS & RECOVERS
STANDS ALONE WELL
STANDS MOMENTARILY
WALKS HOLDING ON FURNITURE
GETS TO SITTING
PULLS SELF TO STAND
STANDS HOLDING ON
SITS WITHOUT SUPPORT
BEAR SOME WEIGHT ON LEGS
PULL TO SIT NO HEAD LAG
ROLLS OVER
STO CHEST UP ARM SUPPORT
SIT-HEAD STEADY
STO-HEAD UP 90°
STO-HEAD UP 45°
STO-LIFTS HEAD
MONTHS
YEARS

Risk for School Failure Using DDST and HSQ Environmental Evaluation

Score	Low (not fostering development)	High (fostering development)
Abnormal	Very high risk	Moderate risk
Questionable	High risk	Moderate risk
Normal	Moderate risk	Low risk

Figure 6

Assessment of a child's home environment appears to be still an emerging field. To date, the best-known screening instrument to determine the home environment's potential for fostering cognitive development is Dr. Betty Caldwell's HOME Scale. Figure 7 illustrates the areas evaluated with the HOME Scale, while Figure 8 presents a more detailed view of one part of the actual interview form.

0-3 HOME Subscales

I. Emotional and Verbal Responsibility of Mother
II. Avoidance of Restriction and Punishment
III. Organization of Physical and Temporal Environment
IV. Provision of Appropriate Play Materials
IV. Opportunities for Variety in Daily Stimulation

Figure 7

V. Maternal Involvement with Child

35. Mother tends to keep child within visual range and to look at him often.
36. Mother "talks" to child while doing her work.
37. Mother consciously encourages developmental advance.
38. Mother invests "maturing" toys with value via her attention.
39. Mother structures child's play periods.
40. Mother provides toys that challenge child to develop new skills.

Figure 8

While the authors have recommended the HOME Scale as a screen, the requirement to spend one to three hours total time to make a home visit preclude it from being used to routinely screen large numbers of children. To overcome this disadvantage, Coons, et al., developed the Home Screening Questionnaire (HSQ) which comes with forms for two age groups.

Home Screening Questionnaire

The two versions of the Home Screening Questionnaire (HSQ) - birth to three years and three to six years - correspond to the HOME Scale developed by Caldwell. Both forms of the HSQ are parent-answered and require approximately 15 minutes to complete. They are both written at the fifth grade reading level, and are appropriate for use with children who reside in circumstances of poverty. Figure 9 depicts a sample of the HSQ.

Questions from HSQ

- How many children's books does your child have of his own?
  _____ 0 too young
  _____ 1 or 2
  _____ 3-9
  _____ 10 or more
- About how often do you take your child to the doctor? ________
- Do you have any friends with children about the same age as your child? YES NO

Figure 9

Recommendations for Screening

While there are a number of viable alternatives in terms of methods of screening, the following represents an approach that has evolved over the past 14 years. Various parts of this approach have been employed throughout the world. The general format is to use a two-phase screen involving the abbreviated DDST followed by the full DDST, and the HSQ followed by a home visit using an interview (such as the HOME Scale). Since the prevalence of school failure is far higher among children residing in homes where the parent(s) has less than a high school education (low SES), a selective screening approach is proposed. This could involve a more intensive screening using the HSQ for high-risk, low SES families. Screening among the higher SES families, where the prevalence of school failure is far less, is more selective and is confined to use of the DDST.

Screening Populations in Which One or More Parents Has Less than a High School Education

The process described (see Figure 10) consists of a first-stage screen with the routine administration of the abbreviated DDST at the following seven age groupings: 3-6, 9-12 and 18-24 months; and three, four, five and six years of age. This first-stage screen can readily be incorporated into the medical evaluation at recommended ages, and only requires about five minutes of additional time. (Children who are suspect on the abbreviated DDST should receive the full DDST immediately on the same occasion. Children who are non-suspect on the full DDST are scheduled for the next periodic re-screen with the abbreviated DDST. Children who are still suspect on the second-stage, complete DDST are scheduled for a diagnostic evaluation.) The second routine screen for all children is that of the home environment with the HSQ, which is scored by a non-professional assistant within a period of about 5-8 minutes.

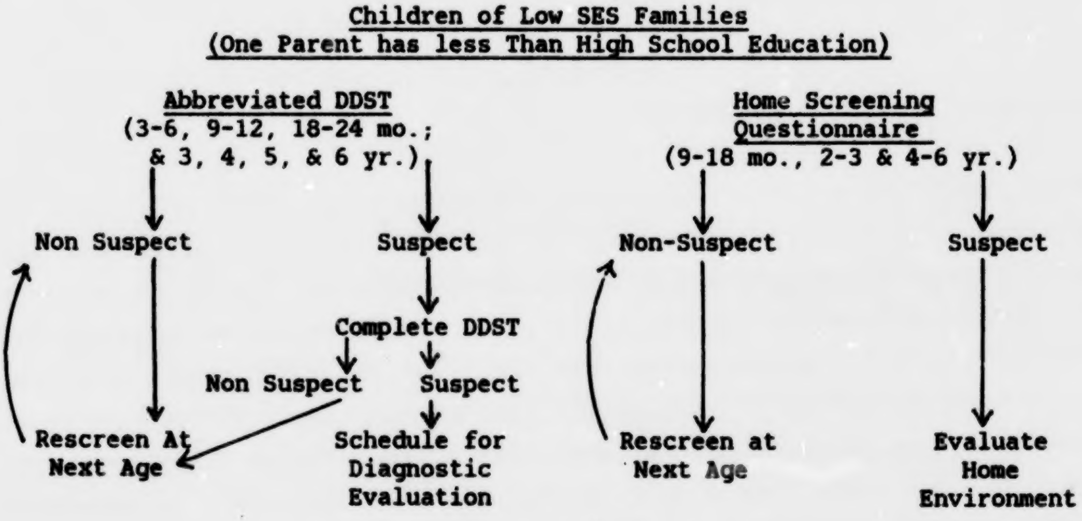
Children of Low SES Families
(One Parent has less Than High School Education)
Abbreviated DDST
(3-6, 9-12, 18-24 mo.; & 3, 4, 5, & 6 yr.)
Non Suspect
Suspect
Complete DDST
Non Suspect
Suspect
Rescreen At Next Age
Schedule for Diagnostic Evaluation
Home Screening Questionnaire
(9-18 mo., 2-3 & 4-6 yr.)
Non-Suspect
Suspect
Rescreen at Next Age
Evaluate Home Environment

Figure 10

Just as all children should undergo routine and periodic re-screening with the abbreviated DDST, they should also undergo periodic re-screening with the HSQ. Between birth and six years, it is recommended that the HSQ be administered three times (9-18 months, 2-3 years and 4-6 years of age). Non-suspect scores should be noted, and re-screening should be performed at the next recommended age. Suspect scores warrant making a home visit to obtain a first-hand view of the home environment. A procedure that may be used as part of such a home evaluation is the HOME Scale. The findings of the home visit should be considered in further planning (such as planning for parent education, parent counselling, or other intervention).

Screening Childhood Populations in Which Both Parents Have at Least a High School Education

While one might prefer to use this Pre Denver Questionnaire (PDQ), the abbreviated DDST, followed by the full DDST, is both an economical process and one that can be easily incorporated into the routine and periodic medical evaluations.

The procedure (see Figure 11) is identical to that for children whose parent(s) has less than a high school education, except the HSQ is used on a selective basis. That is to say, the HSQ is used as part of the diagnostic evaluation.

Children of Middle and High SES Families
(Both Parents Have Graduated from High School)

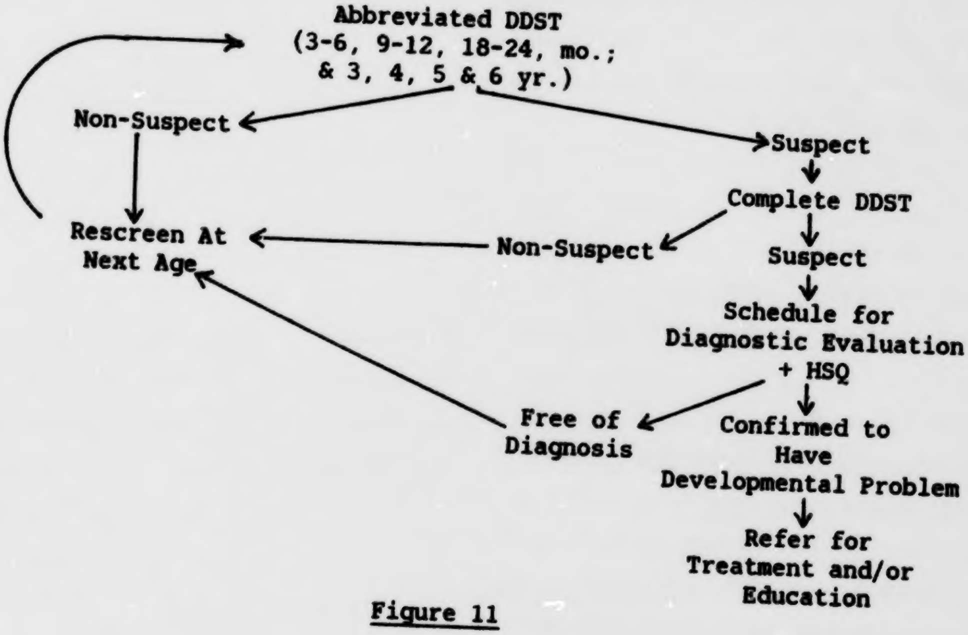
Abbreviated DDST
(3-6, 9-12, 18-24, mo.;
& 3, 4, 5 & 6 yr.)
Non-Suspect
Suspect
Complete DDST
Rescreen At
Next Age
Non-Suspect
Suspect
Schedule for
Diagnostic Evaluation
+ HSQ
Free of
Diagnosis
Confirmed to
Have
Developmental Problem
Refer for
Treatment and/or
Education

Figure 11

Diagnostic Evaluation

Since screening without appropriate follow-up of children who are suspect on screening negates the value of developmental screening, about four years ago we turned our attention to the goal of developing better diagnostic services for handicapped children. The evaluation of developmentally-suspect children by a multi-disciplinary team has, in most countries, been the recommended method for diagnostic appraisal. Indeed, the value of such a team approach cannot be denied; however, there are certain drawbacks to confining ourselves exclusively to its use.

Multi-disciplinary team evaluations, which are quite expensive and time-consuming, are not readily available to children in all communities, and there are simply not enough centres offering this type of evaluation to meet the expected needs of the population. If, in the U.S., all of the currently mandated and recommended screening programs were to be implemented, virtually every child would undergo developmental screening several times before reaching the age of six years. A conservative estimate is that 10% of children being screened require diagnostic evaluation and periodic re-evaluation. Thus, it becomes clear that existing diagnostic facilities are gravely inadequate. It is, therefore, of the utmost urgency that massive nationwide efforts be undertaken to train professionals in diagnostic evaluation.

Recognizing both the urgency and the international need for such training, the Denver group set out to develop a training program that would readily lend itself to replication on a world-wide basis while still recognizing the diversity of situations encountered. The format of instruction utilizes four separate elements: a tutor who is knowledgeable in the subject matter; videotapes to demonstrate cases and evaluation techniques; a student text and time for discussions; and practicum. Subject areas addressed in the training include the following:

1. Medical History and Physical Examination
2. Ophthalmological Evaluation

3. Audiological Evaluation
4. Speech and Language Evaluation
5. Neurological - Neuromotor Evaluation
6. Metabolic Evaluation
7. Genetic Evaluation
8. Psychological Evaluation
9. Abuse and Neglect Evaluation

In addition the clinician learns how to:

1. Integrate diagnostic findings
2. Conduct parent conferences
3. Plan for a child's education
4. Utilize community resources

Training is provided by a tutor, who has special training in child development, who has undergone an orientation, and who has a tutor's guidebook which presents background reading and suggests a format for instruction. Unique features of the training program, which teaches "hands-on" diagnostic skills, includes the semi-tutorial nature of the program, the teaching in small groups, the extensive field-testing of the program and its approval for continuing education credits. Currently, there are 126 tutors for the diagnostic program, including one in almost every state of the United States, and in eight foreign countries. Last year the tutors trained more than 500 physicians; this year it is anticipated that they will train twice that number. Evaluations conducted with the first 220 persons trained in this course demonstrated that they increased their knowledge and perceived competence in each area taught; in addition, more than 95% said they would recommend the course to others.

With the recognition of the importance of training other health professionals to evaluate and treat the infant and pre-school-age handicapped child, the Denver group developed parallel training programs for psychologists, speech pathologists, social workers, foster parents, nurses and others. Currently there are more than 80 lessons available, many of which lend themselves to training specialists in a variety of disciplines. For instance, the lessons on screening can be used by nurses, physicians, psychologists and others. Similarly, the lesson on counselling families can be used by a variety of disciplines. Tables 3 to 6 list a few of the topics presented.

20 PSYCHOLOGY LESSONS

1. Rationale for Assessment
2. Overview of Assessment Techniques
3. Effects of Infant Behaviour on Assessment
4. Techniques of Infant Assessment
5. Assessment of Cognitive Development During Infancy
6. Effects of Pre-schooler's Behaviour on Assessment
7. Assessment of Cognitive Development in the Pre-school Years

Table 3

8 LESSONS FOR SOCIAL WORKERS AND HUMAN SERVICE PROFESSIONALS

1. Jimmy's Parents
2. Introduction to Family Dynamics
3. Family Assessment
4. Case Management
5. Job Stress

Table 4

15 SPEECH AND LANGUAGE LESSONS

1. Preverbal Communication of Children
2. Evaluation of Language Impaired Pre-school Children
3. Oral Reflex Development
4. Therapy for Oral Reflex Problems
5. Oral Motor/Feeding Assessment of Handicapped Children
6. The Physically Handicapped Child-Positioning Techniques for Speech and Language Therapy
7. Hearing Testing of Infants Using Noisemakers

Table 5

9 LESSONS FOR FOSTER PARENTS

1. Introduction to Foster and Adoptive Parenting
2. Issues in Parenting the Special Needs Child
3. Handicapping Conditions and the Development of the Special Needs Child
4. Behavioural Analysis of the Special Needs Child
5. Professional Consultation and the Special Needs Child

Table 6

Currently the Denver group is engaged in developing lessons for parents to use in helping other parents to cope with a handicapped child. Other lessons now being developed pertain to education of the handicapped child.

Summary

In summary, we recommend the following as a cost effective approach to early identification of handicapped children.

1. Use a two-stage screening process as illustrated in Figure 3.
2. As a first stage, administer the abbreviated DDST consisting of only 12 items.
3. Children who are suspect should have the entire DDST completed.
4. Figure 10 illustrates the recommended manner of screening children who come from homes where at least one parent has less than a high school education.
5. Figure 11 illustrates the recommended manner of screening children who come from homes where both parents have at least a high school education.
6. Children who are suspect on screening require a diagnostic evaluation.
7. The combined approach of tutors, teaching in small groups, group discussions, practicums, texts and tutors' guides have proven extremely effective in training a variety of disciplines to perform a basic diagnostic evaluation.
8. Currently there are 150 tutors teaching these lessons through much of the world.

Dr. Thomas Paton, of your city, is a tutor previously oriented to provide training in paediatric screening and diagnosis. Since some of you may be considering how such training might be made available to you, I suggest that you contact Dr. Paton for information about his training plans.

NEW ASPECTS IN THE RECOGNITION AND MANAGEMENT OF CEREBRAL PALSY

Part I - Dr. C. Robertson
Medical Director and Paediatrician
Department of Paediatrics, Glenrose Hospital
Associate Professor of Paediatrics
University of Alberta, Edmonton

We have been asked to submit two recommendations for your consideration. These recommendations come from my daily work rather than directly from the paper I am to present. However, I hope that I can give some rationale to the recommendations in the course of this discussion on Cerebral Palsy. The recommendations are:

1. Documentation of all cases of cerebral palsy with confirmation of the diagnosis and subsequent review of all aspects of the natal and the neonatal period, similar to the reviews done in cases of perinatal mortality. In the province of Alberta, we could anticipate that approximately 100 of the children born this year will have cerebral palsy. Many of these children will be diagnosed as having cerebral palsy through one of the two Neonatal and Perinatal Follow-up Clinics.

2. Public awareness campaigns to focus on healthful pregnancies, warning signs of difficulties and the prevention of prematurity. The aim of this campaign would be to drop the low birthweight rate from 6-7% to less than 5% and to lessen the incidence of birth asphyxia.

The Neonatal Follow-Up Clinic of the Northern and Central Alberta Perinatal Program has been studying the profile of abnormal neurological signs in the neonatal intensive care graduates and how these signs relate to outcome. As the study is not yet complete, we have a cross section of information for children at different ages. For the purpose of outcome statistics at two years of age, the children are listed as normal, mild abnormality, or handicapped - moderate or severe.

"HANDICAPPING CONDITIONS" refers to:

Cerebral Palsy
Visual Impairment →Blind
Neurosensory Hearing Loss - moderate-->profound
Retardation - trainable-->profound
Convulsive Disorders - myoclonic
- recurrent grand mal

Handicaps related to attention disorders, lesser mental handicaps and learning disorders are not considered in this group as part of the significant handicapping conditions. Similarly, strabismus has not been considered as a handicapping condition at the two-year level.

Information is now available on 881 children at age two years.

N.N.I.C.U. GRADUATES
born: 1974 - 1979
follow-up: 2 plus years

	Total Handicaps	C.P.
881 (2 years)	10.9%	8%
498 (3 1/2 years)	11.6%	9.2%

You can see that the total handicap is just over 10%, and cerebral palsy is approximately 8%. Thus, cerebral palsy accounts for approximately 80% of the moderate to severe handicapping conditions. The population, referred to here, excludes those neonatal intensive care graduates with a specific syndrome, or inherited disorders known to be associated with mental or motor impairment.

This presentation will cover only a very small segment of a much larger study related to cerebral palsy, and will focus on the neurological examination at six months or less.

NEUROLOGICAL EXAMINATION

Newborn - admission
- discharge
-->3 - 6 months adjusted age
9 - 12 months adjusted age

As mentioned in last year's symposium, and as has been reported by a number of other authors, an abnormal newborn neurological examination at discharge correlates with subsequent handicaps. In the examination after six months of age, protective, balance and equilibrium reactions can be evaluated. Normalcy or the diagnosis of cerebral palsy can be made much more accurately in the second half of the first year than in the stage of gross motor development, where primitive reflexes are regressing, and where higher functions of balance and equilibrium are not yet emerging. Thus, subsequent reviews of the examination at nine and twelve months will be more accurate than this present review.

This paper discusses the initial neurological examination after the neonatal period and up to six months of age, and compares this initial examination at the Neonatal Follow-Up Clinic with the presence or absence of cerebral palsy at three and a half years.

INITIAL NEUROLOGICAL/DEVELOPMENTAL EXAMINATIONS

(498 children)

vs.

CEREBRAL PALSY/NO CEREBRAL PALSY - Age 3 1/2 Years

(46) (452)

Of the 498 children with complete examinations initially and at age three and a half years, 9.2% or 46 of the children had cerebral palsy.

The cerebral palsy as diagnosed at age three and a half years has been grouped into four main areas as seen here.

CEREBRAL PALSY - TYPES
46 Children

Monoplegia/Hemiplegia	24%
Diplegia	32%
Hypotonia	6%
Quadriplegia	38%

Only 1% of the children seen by three and a half years had any evidence of athetosis, and this has been included in the quadriplegia group. Other than the hypotonia, all of the disorders were spastic.

One of the significant neonatal factors was the classification grouping assigned to the child in the first week of life before the child came to the Neonatal Follow-Up Clinic. There was a greater than 90% follow-up of the children in groups one, two and three.

NEONATAL INTENSIVE CARE GRADUATES - CEREBRAL PALSY
46 Children

	Groups	% C.P.
1.	≤ 1500 grams	14.9
2.	> 1500 grams with C.N.S. problems	12.3
3.	> 1500 grams - ventilated without C.N.S. problems	0.8
4.	> 1500 grams - other (problems requiring admission to intensive care units)	0.8

The overall percentage of children with cerebral palsy is almost 15% in the infants less than 1500 gram birthweight. In the children greater than 1500 grams birthweight, cerebral palsy is highest in those children with birth asphyxia at term. In other graduate studies, the incidence of cerebral palsy was less than 1%.

One of the strengths of this study was to keep the examinations as consistent as possible. All neurological examinations were done by myself, other than during periods of illness, when they were completed by Dr. J. Watt or Dr. R. Shea. The children in this study were born from August of 1974 to December of 1977, and were cared for in either the University or Royal Alexandra Hospital Neonatal Intensive Care Units.

Intervention specifically related to motor or cognitive development, prior to the age of six months, was not given unless the child demonstrated both abnormal neurological signs and delayed development. A format for the neurological examination is outlined in the Glenrose School Hospital, Neonatal Follow-Up, Data Collection and Coding Manual (copyright 1980).

The initial examination included the following:

INITIAL EXAMINATION

≤ 6 months adjusted age

Primitive Reflexes
Head Righting Reactions
"Awareness" - "Excitability"
Spontaneous Motor Activity
Muscle Tone
Gross Motor Development
Deep Tendon Reflexes

The gross motor development was compared to Gesell standards for full-term infants, using the corresponding post-conceptual age.

The reflexes and reactions were analyzed as normal or abnormal for adjusted age for this portion of the study. An example of the outcome results for the "Moro" is given below.

MORO

	Normal	Abnormal
No cerebral palsy	366 - 81%	86 - 19%
Cerebral palsy	21 - 45.7%	25 - 54.3%

An abnormal Moro, ATNR, palmar grasp, placing reaction or absent lateral head righting reaction, if the child was six months of age, were all highly significant for differentiating the child with cerebral palsy from the child without cerebral palsy on the corrected chi square correlation.

REFLEXES AND REACTIONS — **OUTCOME**
≤ 6 months adjusted age — 3 1/2 year

	No C.P.	C.P.	Significance
Abnormal Moro	19%	54.3%	0.0000
Abnormal A.T.N.R. (R)	8.5%	47.8%	0.0000
Abnormal Grasp (R)	7.1%	39.1%	0.0000
Abnormal Placing (R)	20.1%	50%	0.0000
Abnormal Lateral Head Righting (R)	3.5%	30.4%	0.0000

The child's awareness as determined by the same examining physician was as significant as the primitive reflexes.

"AWARENESS" — **OUTCOME**
≤ 6 months adjusted age — 3 1/2 year

	No C.P.	C.P.	Significance
Decreased Sound Awareness	5.2%	21.7%	0.0000
Decreased Visual Awareness	7.9%	39.1%	0.0000
Difficulty to Pacify	6.0%	15.2%	0.0399
Apathy Syndrome	3.4%	15.2%	0.0009
Hyperexcitability Syndrome	2.4%	10.9%	0.0065
Dislikes having Head Touched	2.4%	15.6%	0.0000

Hypertonia and decreased spontaneous movement were also highly significant.

MUSCLE TONE AND MOVEMENT — **OUTCOME**
≤ 6 months adjusted age — 3 1/2 year

	No C.P.	C.P.	Significance
Hypertonia	5.8%	45.7%	0.0000
Hemi syndrome	1.7%	10.9%	0.0011
Abnormal Spontaneous Motor Activity (Prone)	30.2%	75.0%	0.0000

Delayed gross motor development and increased deep tendon reflexes were also highly significant.

MOTOR DEVELOPMENT AND D.T.R.s ≤ 6 months adjusted age		OUTCOME 3 1/2 year	
	No C.P.	C.P.	Significance
Delayed Gross Motor Development	55.5%	75.0%	0.0000
Increased Knee Jerk	17.4%	60.9%	0.0000

In summary, some of the examination was helpful in differentiating the cerebral palsy from the non-cerebral palsy children. Primary walking, positive support reaction and hypotonia were not significant in differentiating the two groups in this population. Remember that the age of the child was after the newborn period and under six months, and that this was already an at-risk population.

NOT DIFFERENTIATING C.P./NO C.P.
- in this N.N.I.C.U. population
- at this age

Primary Walking
Positive Support Reaction
Hypotonia

Hypotonia was not significant, as in this population it was a global characteristic and found in 80% of the children who had subsequently had no cerebral palsy.

Differentiating tests were grouped according to the frequency that the test was abnormal in the cerebral palsy group. The Moro, Asymmetrical Tonic Neck Reflex, Palmar Grasp, Placing and Hypertonia all differentiated cerebral palsy from non-cerebral palsy. However, each of these, when analyzed in isolation, was normal in up to one half of the cases of cerebral palsy.

DIFFERENTIATING C.P./NO C.P.

(40 - 50% of C.P. were not abnormal)

- Moro
- ATNR
- Grasp
- Placing
- Hypertonia

Decreased spontaneous motor activity and delayed gross motor development were very significant as differentiating cerebral palsy from the non-cerebral palsy group, and in each 75% of the cerebral palsy group were abnormal at six months or less.

DIFFERENTIATING C.P./NO C.P.

(25% of C.P. were not abnormal)

- Spontaneous Motor Activity
- Gross Motor Development

The remainder of the areas tested differentiated between cerebral palsy and no cerebral palsy, but the percentage of abnormality was low in each group. For example, the child's dislike of having the head touched was present in the non-cerebral palsy group in only 2.4% of children. In the cerebral palsy group, it was present in 15.6% of children.

DIFFERENTIATING C.P./NO C.P.

(% abnormality C.P. - 10→30)

(% abnormality No C.P. - 1→4)

- Lateral Head Righting
- Hemi Syndrome
- Apathy Syndrome
- Hyperexcitability Syndrome
- Dislikes Having Head Touched

A factor analysis is now being done to look at each area more closely and to determine which constellation of factors may be most useful under the age of six months to predict the diagnosis of cerebral palsy. May I suggest from the information given that the gross motor development and an observation of the quality and quantity of spontaneous motor activity could be used by primary health care workers more than at present. Further evaluation will be done in an attempt to correlate the early abnormalities with the type and severity of cerebral palsy, and to correlate those factors which were abnormal, but where the child did not have cerebral palsy with the outcome of that child at three and a half years. It is anticipated that these abnormalities will be significant for other developmental difficulties at three and a half years.

I shall turn now to the question of management of cerebral palsy in a general sense. A great deal can be done by the primary health care worker to assist the cerebral palsy child and his or her family, both before the diagnosis is made and subsequently.

EARLY INTERVENTION - GENERAL

Immediate - before diagnosis
- Family
- Feeding
- Handling
- Sensory Input

Some of the early therapy begins prior to the birth of the child in terms of the attitude of the parents and community to the unborn child. However, I will not discuss this at this point. Once the child has demonstrated a delay in gross motor development, difficulties with feeding, poor quality of movement; whether or not a diagnosis of cerebral palsy has been made, the prime factor in assistance of this family is the support of the family. Much of the so-called "family support" centres around helping the parents to parent the child. By this, I mean aid with neuromuscular feeding problems, suggestions to decrease the central nervous system irritability which one often sees, and additional understanding and general health care for the child who has been ill. Discussions of the pregnancy and neonatal problems may also be of benefit. Assistance in the physical handling of the infant so that this child becomes accustomed to cuddling, suspension in space, movement in space and tactile stimulation, may be extremely beneficial at a later date. Not only will this be helpful to the treating therapist in her physiotherapy for the child, but it also aids the child in reaching his or her potential rather than adding to the child's limitation by further learned fear of movement and dislike of touch. Sensory input, as done by an attentive mother with a healthy baby, is also beneficial, but may be difficult in an irritable infant with decreased awareness. Much of the key for the early intervention, i.e., prior to six months, could come under the phrase "good mothering" with a strong bond built between the child and his or her parents.

Once the diagnosis of cerebral palsy has been made, the requirement for family support cannot be over-emphasized. This support is more effective if the counsellor has knowledge of the projected outcome of the child and is prepared to answer parent questions in detail. Part of this counselling involves an awareness of other handicapping conditions which should not be overlooked.

CEREBRAL PALSY

- Other major handicaps

	%
Cerebral Palsy only	73.3
One additional handicap	16.9
Two additional handicaps	5.6
Three additional handicaps	4.2

(attentional and learning disorders - NOT included)

Other moderate to severe handicaps occurred in greater than one quarter of children with cerebral palsy in our study. Strabismus was not included as a significant handicapping disorder in this report. The majority of cerebral palsy children will have attentional disorders and perceptual difficulties which will interfere with learning. A comparison of the cerebral palsy to the non-cerebral palsy group with regard to learning is part of our long-term follow-up study.

When we talk about early intervention for the child with cerebral palsy, we immediately think of physiotherapy. However, in addition to the specific therapy, the child requires "general intervention".

EARLY INTERVENTION - GENERAL

Ongoing - "Goals"
Cognitive Skills
Central Processing
Maturation
Total Development

General intervention should be given with specific goals for short-term and long-term achievements related to all areas of development. Although the initial intervention is "motor", it is important that these children receive detailed pre-school psycho-educational and occupational therapy assessments and early assistance for any particular central processing and fine motor difficulties that may be present. From an early age, the child should be encouraged to be self-reliant where possible, and should be encouraged to be as emotionally independent and socially mature as his or her non-physically handicapped peers with similar mental ability.

Once the diagnosis of cerebral palsy is made, the therapy is very specific and is, increasingly, inter-disciplinary.

EARLY INTERVENTION - SPECIFIC

Highly Specialized
Inter-disciplinary
Re-assessments

Initial: e.g.
- primitive reflex integration
- improvement of quality of movement and tone
- enhancement of righting reactions, stability, balance, and equilibrium
- enhancement of communication

Physiotherapy has changed greatly in recent years and through the efforts of highly trained therapists some of our young cerebral palsied children can reach their motor potential which, I believe, is unlikely without physiotherapy intervention. The areas of stability and balance and equilibrium are now receiving increased interest in the treatment of cerebral palsy, as increasing knowledge is obtained about the development of balance and equilibrium in healthy children. Physiotherapy for the child with cerebral palsy is highly specialized, and, we believe, requires a considerable experience with various types of cerebral palsy at a young age to have a complete understanding of the effect of the disorder on the individual child and the types of intervention required to help the child to reach his or her potential. Communication skills should be started very early and reinforced. This is particularly important for the severely handicapped child, and this is also a highly specialized area at this time.

THE EARLY MANAGEMENT OF GROSS MOTOR DIFFICULTIES IN CEREBRAL PALSIED CHILDREN

Part 2 - Dr. J. Watt
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The management of a cerebral palsied child is best carried out by a multidisciplinary team. This is especially so in younger children affected by the condition. It is obviously impossible for me to discuss the overall management of young cerebral palsied children within such a short period of time. I shall therefore concentrate on highlighting some of the important points in the early management of gross motor difficulties in these children.

In managing the motor difficulties in a newly diagnosed young cerebral palsied child, three aspects deserve special attention. These are: on-going evaluation of the child, early prognostication of motor function and early treatment programs and modalities.

On-going Evaluation

Even though cerebral palsy is caused by a non-progressive lesion to the immature brain, the clinical picture is not unchanging. It is important to follow the child on an on-going basis to observe the changing clinical signs and symptoms in order to design a sensible management program and set a realistic goal. I feel that during the first three years of life, these children should be followed and assessed every three to six months by the treatment team and the physicians to detect and document changing tone and reflexes. Not uncommonly, the clinical type of cerebral palsy cannot be diagnosed accurately until after one year of age. This is especially so with reference to choreoathetotic cerebral palsy. This, of course, will completely alter the plan of management. Joint ranges, especially those around the hips, should be documented every three months, and, should there be any significant change, x-rays should be obtained to rule out correctable orthopaedic deformities. The developmental milestones attained are important in the prognostication of motor functions, and obviously it will decide the next therapeutic approach. Evaluation of the effect of treatment is often very difficult, especially when it has to be done objectively. It often helps if the child is assessed by the physician or another therapist not involved in the treatment of the child.

Early Prognostication of Motor Function

The most common question the parents ask about gross motor function of a cerebral palsied child is, "Will my child walk?" There are a few studies in literature which I have found useful in answering this question. Bleck(1) advocated the use of primitive reflexes in prognostication of locomotor function. A scoring system is designed whereby the persistence of each abnormal primitive reflex is given a score of one. This applies to the asymmetrical tonic neck reflex, symmetrical tonic neck reflex, Moro reflex, neck righting reflex and extensor thrust. In addition, failure of appearance of the foot placement reaction and the parachute reaction is each given a score of one. The scores are added together, giving a maximum score of seven. In a group of 73 children followed at his centre, he found a score of zero indicated a good prognosis, a score of one indicated a guarded prognosis and a score of more than two indicated a poor prognosis. The accuracy of prediction was 94.5% reliable when done after the age of one.

In another more detailed study of 233 children, Molnar and Gordon(2) examined children after the age of two. The prognosis of locomotion depends on the type of cerebral palsy. They found that all hemiparetic and ataxic cerebral palsied children could walk. For the other types of cerebral palsy, the prognosis depended on whether or not they could sit by the age of two. For those who could sit by the age of two, all of them eventually could walk. For those not able to sit independently by the age of two, two further groups were identified. Again, they found that the persistence of primitive reflexes including the Moro, ATNR, STNR, tonic labyrinth, positive supporting and extensor posturing reflexes indicated poor prognosis. The group that could suppress the primitive reflexes between 18 and 24 months had favourable prognosis. The above studies are brief guidelines, but they are not hard and fast rules. When parents of cerebral palsied children do ask the question of "will my child walk?", most of them have come to the conclusion themselves that the child will probably not be able to walk in the future. I found that most parents are actually relieved when they are given a honest answer in a "matter of fact" and sympathetic way. It is crucial at that point to suggest an alternative means of mobility, and emphasize that walking is just one method of mobility. On the other hand, if the treatment team realizes that the child is not going to be a walker, while the parents have not suspected that to be so, the treating therapist should educate and prepare the parents to face this reality in a gradual way. It is worthwhile to demonstrate to the parents in therapy how impractical it is for the child to walk. If this is done, it will avoid the embarrassment to the treating therapist and trauma to the parents when they are told of the prognosis by another party, very often a non-professional person. Most of the locomotor prognostication studies indicate that if a child fails to walk independently by the age of seven, there is really no chance of walking thereafter.

Treatment

The goal of treatment is not to strive for neurological cure, it is to improve function. In the early treatment of gross motor difficulties, family counselling, early intervention, physiotherapy and occupational therapy are the corner stones of the total management program. However, there are other physical modalities that will serve as adjuncts to the conventional therapy. I shall spend the rest of the time discussing some of these.

1. Seating. Seating has been more and more recognized as an important aspect of rehabilitation of cerebral palsied children. According to Rang(3), the goals of seating include comfort, improved function, alleviation of deformity, easier transportation and to make life easier for the parents.

   For the younger children, the group which we are interested in, it encourages head control, it helps to free the upper limbs for functional use, it encourages the development of independent sitting balance, it makes feeding and swallowing much easier and it discourages abnormal sitting posture due to abnormal tone or primitive reflexes.

2. Inhibitive Casting. Spastic cerebral palsied children are usually toe walkers when they first start to walk around furniture, and later on independently. This makes their walking base much smaller, and encourages total extensor thrusting. Domisse(4) advocated inhibitive casting as early as 1964. The use of a below knee cast to maintain reflex inhibiting postures to control tone was found to be effective in controlling equinus posturing. The casts were applied with the ankle dorsiflexed, the foot supinated and digits forcibly dorsiflexed.

   Mott and Sussman(5) in 1974 revived the use of these inhibitive casts. They found them effective in decreasing tone and improving ambulatory status. The hypothesis put forward included the fact that extending the toes inhibits plantar grasp response, thereby contributing to tone reduction. The firm fixation of an unstable foot and ankle provides a stable base of support, which further reduces tone and facilitates mobility of the knees, hips and trunk. The prevention of ankle plantar-flexion inhibits extensor thrust. They also felt that the cast provides full control of foot and ankle, and frees the therapist to concentrate on other areas of the body.

   We have started doing this sort of inhibitive casting in the early part of 1980. We are in the process of doing a study on cerebral palsied children who can at least do supporting standing. The problem is that dynamic equinus posturing in the passive dorsiflexion of the ankle should be possible past 5°. The child is seen by a physician and a physiotherapist who are not involved in the treatment of the child before casting. The casts are put on for three weeks during which the child receives three times weekly gait training. This is because the casts are just adjuncts to physiotherapy. After coming out of the cast, children are allowed to walk for around five weeks, and they will be seen again by the physician and physiotherapist.

3. Orthosis. A word about orthoses for controlling equinus posturing. In most centres, orthopaedic shoes, traditional double up right braces, long leg braces and full control braces are gradually being phased out. This is mainly because of their weight, poor cosmesis, inadequate

control and covering up of deformities. We have found polypropylene ankle foot orthoses to be the most useful, especially with children who have had inhibitive casting to correct some of the dynamic contractures. Polypropylene KAFO's are used occasionally for children with severe recurvatum, usually secondary to lengthening of the hamstrings at surgery. Orthoses are useless in dealing with static fixed contractures.

4. Surgery. During the first three years of life, surgery is seldom indicated. Hoffer(6) in 1980 said that they seldom perform surgical procedures during the first three year dynamic neurologic period.

   However, the only exception is surgery of the hips. Early adductor tenotomies prevent hip subluxation, stop scissoring and prevent pelvic obliquity which will eventually result in wind swept posturing. When the hips cannot be abducted to less than 30°, adductor surgery should be done. X-rays of the hips should be taken at least every six months when the hips are tight. Any sign of subluxation should be tackled immediately. Preventing subluxation of the hips is a much simpler and effective procedure than to remedy a dislocated hip.

   On the other hand, there are few reasons to perform heel cord lengthenings under the age of five years because patterns of walking are not well established(7). This is the period for physical therapy, possible application of an ankle foot orthosis and tincture of time. There is a misconception that orthopaedic deformities interfere with the initial attainment of gait. This is not so. Orthopaedic deformities will worsen a gait pattern, but they will not stop the initial attainment of gait.

5. Rehabilitation Engineering. Rehabilitation Engineering has a lot to offer to the total rehabilitation of the cerebral palsied child. Biofeedback devices, such as positional control devices and communication devices, are helpful in enlarging the scope of the child's life. Various sophisticated mobility aids and control systems help the child get around. Gait laboratory helps to objectively measure the effect of

treatment and surgery. Unfortunately, in Alberta, Rehabilitation Engineering is not in existence. This is one area that we should really improve on. In other centres, most people have felt that Rehabilitation Engineering will become the most important modality in the management of cerebral palsied children in the future. I hope that the Alberta Government can start to look into this area, so that the cerebral palsied children of Alberta can benefit from this new and exciting treatment modality.

In conclusion, I would like to bring the message across that all is not lost when a handicapping condition cannot be prevented. There are a lot of things that we can do to improve the quality of life of the handicapped child and the family.

May I, in this International Year of Disabled Persons, suggest to the Alberta Government to spend as much effort in the support and care of the disabled child as they have, in the prevention of handicapping conditions. A symposium on the management of developmental handicapping conditions is in order. Thank you.

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EARLY IDENTIFICATION OF CHILDREN WITH COMMUNICATION DISORDERS

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Introduction

I am pleased to have this opportunity to speak with you today about communication disorders and early intervention. During my presentation, you will find me using the words 'speech and language' and 'communication' somewhat interchangeably. This is to remind you that speech pathologists are concerned with all aspects of an individual's ability to communicate on all levels of communication. Intervention may prove necessary at any time in an individual's life, from the moment non-verbal messages begin passing between an infant and mother during feeding until, as an adult, speech is fluently articulated and is grammatically and semantically correct, and appropriate in all situations. If the acquisition of conventional speech and language skills is not an achievable goal with an individual, then providing the most functional communication system possible becomes our goal.

Over the past five years, I have assessed and subsequently followed the speech, language and feeding development of many children in the six month to three and a half year age range. This has largely been due to my involvement with the Neonatal Follow-up Clinic at the Glenrose Hospital. As you may know, the clinic was established in 1974 as part of the Northern and Central Alberta Perinatal Program. It was set up as a longitudinal research study of children with selected perinatal problems who were cared for in the Neonatal Intensive Care Units at the Royal Alexandra Hospital and University of Alberta Hospital in Edmonton. Children being followed include those weighing 1500 grams or less at birth, those weighing more than 1500 grams at birth with central nervous system conditions complicating their course, 50% of children weighing more than 1500 grams at birth and requiring ventilation, and children weighing more than 1500 grams at birth with specific problems of concern to the neonatologist, but not fitting into the previously defined groups. Over 1,000 children are being followed from birth to age eight through this study. My involvement has been primarily with the children from eighteen months of age to three and a half years of age. I also see a number of children earlier than eighteen months at the request of a parent or clinic team member. What this experience has exposed me to is a population of children who are potentially 'at risk' for manifesting a variety of problems, including those in the area of communication.

The National Institute of Neurological and Communicative Disorders and Stroke(1) in the United States collected speech, language and hearing data as part of their Collaborative Perinatal Project. This project registered pregnant women from January, 1959 to December, 1965. The children were followed from the pre-natal period to eight years of age. Variables relating to pregnancy, labour and delivery, family characteristics, and physical, mental and behavioural characteristics of the children were studied to provide clues to the etiology of communication disorders and to uncover findings which might be clinically applicable as well as predictive of outcome. 27,559 children were included in the study of speech, language and hearing data. Perinatal factors that were found to be associated with less favourable speech, language and hearing outcomes included birthweight less than 2500 grams, microcephaly and abnormal neurological findings. Physical factors found to be associated with less favourable speech, language and hearing included high parity, maternal mental retardation, leanness, and male gender. With respect to articulation specifically, a summary of the results of the study concluded that social variables are more strongly related to articulation growth than physical factors, except where there are clearly relevant organic deficiencies as in cerebral palsy, cleft palate and mental retardation.

Eugene McDonald(2), after a review of the literature, stated that "conditions frequently associated with brain damage such as maternal illness during pregnancies, difficult delivery, low Apgar scores and atypical neonatal signs, should cue a physician to alert a specialist in early speech development to follow the infant".

A large number of children are included in the descriptions given of children potentially at risk for developing communication disorders. At the present time, it is not possible to have speech pathologists following all these children. In fact, it should not be necessary, if a number of factors are present to ensure that infants or children are referred for follow-up when appropriate. Firstly, individuals, such as primary care physicians and nurses, need to be aware of the importance of early referral and intervention with children and their families, when factors are present, which place the child at risk for developing adequate speech and language skills. Secondly, these same individuals need to know the kinds of early problems a speech pathologist can and should be helping them with. Finally, primary care health professionals need to be familiar with the diagnostic signs that suggest that an infant or young child is having difficulty with pre-speech skills, or is falling significantly behind in comparison with normal children such that he is at risk for developing an adequate communication system.

Communication Disorder Predictors

I am going to discuss the issue of diagnostic signs first, and return to the other issues later. Obviously, you are not going to refer every child who is slightly behind in pre-speech or language skills to a speech pathologist. We seem to have few problems getting children referred who are showing general comprehension delays, although these children could probably be referred earlier in some cases. Comprehension or receptive language delays rarely occur in isolation. There are usually accompanying delays in expressive language. The child may be engaging in frequent vocal play but showing little verbal or non-verbal interactive communication. We may be alerted by poor attending to people or differences in the way the child interacts with toys. There are few good assessment tools for assessing a child's language before he reaches the age of about three years, and those we have are limited by the amount of co-operation we get from the child. The best way to be sure you are getting accurate information about young children's language is to observe them in interactions with their parents, and with toys they enjoy, ask very specific questions of the parents, and then compare your findings with the normative data. This may sound very common sense and obvious to you, but I frequently return to the normative data to remind myself what a child of a particular age should be doing. It is too easy to fool ourselves into thinking that a child is not doing "too badly" when in fact he is significantly behind his peers.

Going back to the question of assessment tools for a moment, it is interesting to note that, in the Collaborative Project in the United States I earlier referred to, results on the Bayley Scales administered at eight months were not found to be predictive of future language problems. However, when the items were analyzed individually, children who failed the items 'vocalize to a social stimulus' and 'vocalizes two syllables' had a 3-4 times higher chance of later poor language development.

My biggest diagnostic headache has been the child who is understanding what he should be for his age, and is communicating well non-verbally, but is behind in terms of the vocal output he should have for his age. He may or may not have a behaviour problem. Most physicians have a first line of defense with this type of child. For example, a mother brings a two year old to see him, because the child only says the words mama, oh oh, bye bye, and wow. The physician says "don't worry", and suggests that the mother start expecting the child to ask for things with words. He then says, "let's give it another six months, and if he's still behind, we'll refer". Often, the child returns in six months and is doing fine. The two things that amazed me the most during my first year in the Neonatal Follow-up Clinic were the range of expressive speech and language skills I observed in children between the ages of 18 and 30 months, and the rapidity with which the skills could improve during that period. The difficult issue was predicting which of the children might not improve. This is where one's diagnostic skills are particularly challenged.

Remember that speech is a complicated process, brought about by activities of a large portion of the body structure - activities that involve the use of large and small muscle groups in both sequential and overlapping action. The motor act of speech requires rapid production of all the vowels and consonants present in the English langauge in an infinite number of combinations to produce words. The production of one sound requires the co-ordination of respiration, phonation, resonance and the movement of the jaw, lips, tongue and palate. The normally developing infant begins to practise sounds and their combinations in the babbling stage. He also begins to practise the intonation patterns of our language at an early age. When you are trying to decide if a child's speech and expressive language is going to improve without intervention, really look at what the child is able to do with respect to the different components that are necessary for producing speech and expressive language. Ask yourself these questions:

1. Is the child using his voice to get attention? Does he know that the voice communicates?

2. Is the child producing several consonant-vowel combinations (several syllables) on one exhalation?

3. Is the child using a variety of different consonants and vowels?

4. Is the child not only using reduplicated babbling (e.g., bababa) but changing both the consonant and the vowel in one utterance (e.g., badido)?

5. Is the child practising inflections while producing several consonant-vowel syllables on a single exhalation?

6. Is the child using the same combination of sound consistently in association with referent, that is, using a word?

7. Is the child using a clear voice quality in his vocalizations?

These are all skills that a child should be using by the time he is 12 months old, earlier in several instances. He should be vocalizing to get attention, producing several syllables on one exhalation, be using a variety of vowels and consonants across utterances and within utterances, and be imitating simple sound patterns. Eugene McDonald's(2) chapter in Nonspeech Language and Communication Analysis and Intervention provides an excellent breakdown of the development of differentiated vocalization from birth to 12 months.

If you feel, after exploring a child's vocal behaviour, that there are some concerns in the area, question further to determine if there were any early indicators of lack of control in the oral or respiratory systems.

1. Is there any history of feeding difficulties; arhythmic or weak sucking; lack of co-ordination of sucking, swallowing or breathing; persistence of choking when new food textures were introduced; difficulty keeping food in the mouth?

2. Did the child continue to drool excessively past the age of nine months even when he was not cutting teeth?

3. Is the child past the age of 12 months and still now showing the rotary jaw movements or lateral tongue movements in transferring food to the sides of the mouth?

4. Is the child's face asymmetrical, or does the tone appear low as indicated by drooling and an open mouth posture?

5. Is there a history of feeding by non-oral means, prolonged intubation, an atypical cry, or respiratory dysfunction?

There is no firm evidence that any of the factors just noted cause speech and language problems, or are predictive of these problems. However, clinical experience suggests that the same children who have difficulties in non-speech use of the oral mechanism often have difficulties when the same system needs to be used for speech. There is an excellent paper on "Parellel Patterns of Oral Motor Development in Feeding and Speech Production",(3) if you are interested in further information on this topic.

Some Comments on Early Treatment

I would like to go back now and discuss some of the early problems related to communication with which a speech pathologist should be able to assist the primary care health professional. I do not have time to go into all of them, so I will just touch on three problems I have particularly come in contact with in conjunction with my work at the Glenrose Hospital.

The first problem is that of the infant with persistent feeding problems. These problems effect the infant's ability to get adequate nutrition in a reasonable amount of time. A speech pathologist or occupational therapist, with special training or experience in the area of feeding, should be available to help the mother with facilitating the best feeding skills possible before abnormal patterns are established. Speech movements do not appear to evolve from swallowing, sucking and chewing, but it does appear that if a child cannot develop the patterns of neuromuscular activity necessary for vegetative function, it is unlikely he will develop the patterns required for speech. Thus, the early involvement with feeding will allow the speech pathologist a chance to observe the infant's pre-speech development, and offer suggestions for the stimulation of both pre-speech and communication skills before the family's methods of interaction with the baby become established.

There is a second similar problem with which speech pathologists rarely seem to become involved. That is the problem of the infant who is fed through a nasogastric tube, oral-gastric tube, or gastrostomy for a prolonged period of time. I recently attended a workshop on pre-speech intervention given by Suzanne Evans Morris from the Curative Rehabilitation Centre in Milwaukee, Wisconsin. She felt it was imperative that these children receive an extensive amount of oral stimulation, and that significant gains in pre-speech and speech development often result. Without it, the prognosis for speech development in children with neurological disorders is reportedly often very poor.

The third area where I feel speech pathologists could be getting more involved is in cases where infants present with chronic dysphonia or aphonia. This may develop following prolonged intubation or as a result of the left recurrent laryngeal nerve being damaged during a patent ductus arteriosis repair. These infants are usually seen by an ear, nose and throat specialist for a laryngeal examination, and their parents told that the voice will probably improve gradually. The parents are often concerned about the implications of the problem with respect to speech development. One session with a speech pathologist who has experience in the area can usually provide them with reassurance as to why the problem should not affect speech development. The importance of continuing to talk to the child and engaging in sound play is discussed. The gradual improvements the parents will hear can be outlined, so that they know when to contact the E.N.T. and/or speech pathologist if the voice is not improving. The parents are free to call the therapist for further information if they have questions or concerns. This is often a level of service that the E.N.T. does not have the time to provide.

Referral

I would like to finish my discussion with some comments about referral and subsequent intervention with young children. I would rather assess a child who has normal speech and language than miss a child who has communication disorder or delay. If you are in doubt about whether to refer a particular child, refer, and the speech pathologist can determine whether or not intervention is necessary and what kind of intervention would be the most appropriate.

In the case where you have a child with a delay or problem that appears mild, and your investigation of the warning signs I have discussed reveals nothing of particular concern, offer the parents some suggestions verbally or through language stimulation pamphlets.(4,5,6,7) Suggest a time frame to the parent within which she could see specified gains. If the gains do not occur, then refer on. If the parent is very concerned and does not want to wait, refer right away.

An assessment by a speech pathologist, with subsequent discussion, can result in the easing of a parent's concerns, if there is little cause for the concern. Suggestions can be provided that often make the parent feel they can do something about the slight delay that is present. If concerns surface about the way the parent is handling the stimulation of speech and language skills, suggestions can be given that are appropriate to the particular problems of the child and parent. This in turn can facilitate communication in an atmosphere in which there is as good a chance of success as possible.

If the problem persists, the initial assessment information can be used as baseline data for comparison.

In case of more significant problems, again an early assessment provides baseline data for future comparison, even if a differential diagnosis cannot be made right away. The parent can become involved in home observations to help both the therapist and family discover exactly where the child is functioning and help each to be realistic. The therapist can begin developing rapport with the family, and help them develop an awareness of the problem to the point where they can start to positively work to alleviate the problem as soon as possible. With the child who has poor potential for speech, or one whose speech will probably develop late, emphasis of early intervention can be placed on developing an environment where non-speech communication is encouraged. If, after early referral, it appears that the problem is affecting more than communication, the child can be referred for further assessment. It may prove that a general intervention program is more appropriate than individual speech/language/communication treatment.

An extremely important reason for early referral is the length of speech pathology waiting lists in many parts of the country. Waits of one year are common with waits of three years not unheard of even for children with significant problems. It is easy to take a name off a waiting list if the problem is resolved, but difficult to move a name up if there are 50 names in front of it.

Recommendations

I feel it would be optimal if there were enough speech pathologists to provide both consultation and direct intervention with children as early as a problem has been identified, or is suspected.

When dealing with infants and young children, intervention can take many forms other than one to one treatment in the speech pathologist's office. These would include:

A) an assessment where counselling and suggestions are given and no further involvement is needed;

B) an assessment and periodic review to measure progress and/or provide suggestions;

C) acting as a consultant to another health care professional already involved in the case;

D) going into the home to help a family implement feeding or communication carry-over suggestions.

In closing, I have two recommendations I would like to offer to this symposium.

1. That primary health care professionals refer children to speech pathologists as soon as a problem has been identified that may inhibit pre-speech, speech, language, or communication development, or when a parent indicates genuine concern.

2. That the number of speech pathology positions in centres serving pre-school populations be increased so that early intervention, and, in some cases, prevention of more severe communication disabilities becomes a reality.

It may be necessary to provide additional training to some speech pathologists to help them deal with the specific disorders of infants and young children. For the present, you may need to seek out the expertise if you have an infant with a special problem. But be aware that that expertise is available, and that children with communication delays and disorders have a right to receive it.

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THE HIGH RISK CONCEPT REVISITED

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As part of its comprehensive pre-school health surveillance program the province of Alberta has, for the past few years, utilized the high risk concept to identify handicapped children. I have been asked to comment on the use of the high risk concept, even though I have not personally employed it. During my brief presentation it is my intention to review its history, to compare it to other screening as a method of early identification, to make some comments regarding the Alberta program, and to give you in the audience, who have had first-hand experience with it, an opportunity to discuss aspects of the risk register. In summary, it is the shared aim of Dr. Tom Paton and myself to prompt you to think critically of various case finding approaches.

History

The high risk concept seems to have started in 1957 when Dr. Fisch, a British otolaryngologist who was interested in the early identification of deaf children, proposed a new approach to the early identification of congenital deafness. His approach was based upon several assumptions:

1. early diagnosis and treatment of hearing loss assures best results;
2. routine and periodic screening of all children is not feasible, due to lack of sufficient staff, equipment and facilities;
3. rapid, imprecise screening may lead to under-referrals, and may therefore delay treatment.

He therefore suggested careful examination of a few children who, on the basis of the medical history, had a high probability of having a hearing loss. The factors he proposed were:

1. genetic
2. pre-maturity, and
3. peri- and post-natal factors.

He suggested that a co-operative effort be undertaken between physicians, health departments and practitioners to identify these children before the usual time.

Four years later (in 1961), Dr. Lindon suggested that the approach be broadened to include children with visual and motor defects, such as cerebral palsy, since these conditions would also benefit from early diagnosis and treatment. He suggested concentrating efforts on a few "at risk" infants since he was of the opinion that 20% of the population would contain 70% of the handicapped children. His persuasive arguments culminated in a proposal that the National Health Service establish an "At Risk Register".

In the following year, Dr. Sheridan amplified upon the recommendations of Fisch and Lindon in her well-known article in a Ministry of Health publication. She, too, estimated that about 20% of children would be listed on such registers. In the following year, the Ministry of Health asked all health authorities to compile "at risk registers" and to collect such data.

In subsequent years, data began to come in and the results were not as anticipated. For instance:

1. in Harlow, among 500 consecutive deliveries, 83% of children were placed on the register,
2. few of the children on the registers turned out to have problems,
3. children not on the registers were neglected in terms of adequate health surveillance,
4. the registers caused undue anxiety to parents whose children were on the register,
5. the time and expense required to carry out such registers are exceeding previous expectations,

6. many handicapped children were not on the register, and
7. there was a danger of forgetting that a child with one handicap may be at risk for another.

Problems with Risk Registers

What went wrong? Was it the concept or the way in which the program had beem implemented? It is my opinion that both were at fault.

In retrospect, there were five major errors in the concept. These were:

1. Failure to explicitly state what handicapping conditions will be identified.
2. Failure to marshall data that the conditions can be identified at the time of birth.
3. Failure to assure that the problem being sought is sufficiently prevalent to justify the effort.
4. Failure to assure that the identification methods are valid or accurate.
5. Failure to assure that the condition selected can be prevented or ameliorated if diagnosed and treated prior to the usual time.

Let us look at each of these in more detail.

1. Failure to Explicitly State What Handicapping Conditions Will be Identified

Unless one states in measurable terms what one hopes to identify and prevent, it is virtually impossible to determine the efficacy of the case finding methods. The various writings about the use of the at risk registers state that they are designed to detect cerebral palsy, deafness and other handicapping conditions. They seem to include all physical, mental and emotional defects likely to affect the growth and development of the child. Not only did the health authorities fail to specify in measurable terms what was to be identified, but they also committed an additional error of telling each health authority to develop their own risk lists, which in turn were to identify the ill-defined handicapped. It is a tragedy that the zeal towards prevention of handicapping conditions blinded the architects of the programs. They not only failed to specify what they were trying to identify, but also in suggesting to local authorities that lacked adequate knowledge and experience in test construction to use whatever they desired to identify the so-called handicapped. An analogy would be to blindfold a person, lead him or her into a dark room and tell the person to hit an unseen target of unknown size with whatever they considered appropriate. Naturally such an effort is doomed to failure.

2. Failure to Marshall Data that the Conditions can be Identified at the Time of Birth

Another fault of the risk approach is its simplistic view of how handicapping conditions develop. This view suggests a simple cause may produce a predictable outcome. Thus, it was suggested that anoxia at the time of birth would cause cerebral palsy. While this is true in some cases, many anoxic newborns never develop cerebral palsy. Similarly mental retardation, especially the cultural familial variety which constitutes 75% of the cases, is considered to evolve as a result of a complex interaction of genetic and environmental factors. Not all of these variables are identifiable at the time of birth. Unfortunately, the pathogenesis of most handicapping conditions is not sufficiently understood to know which factors - and the degree to which each contributes to future handicaps. Certainly it is fallacious to think that all of the factors can be predicted on the basis of a health survey at one point in time such as at birth.

3. Failure to Assure that the Problem Being Sought is Sufficiently Prevalent to Justify the Effort

Galen and Gambino have demonstrated that when screening for a disease, the less prevalent the condition, the higher the number of false positives. Similarly, Fleeson, et al., have demonstrated that the rarer the disease or handicap, the higher the number of false positives. For instance, a group of international specialists meeting in Nova

Scotia a few years ago, recommended that every newborn be reviewed for the presence of 1-5 risk factors for congenital hearing loss. This condition has a prevalence of one per thousand. Use of these few risk factors generates a suspect rate of 15-20%, with a 99% false positive rate. One reason for the high number of false positives is the low prevalence of the condition. It has also been demonstrated that the greater the number of conditions one is seeking to identify, the less chance the individual has of coming out non-suspect on each of the procedures or tests. For instance, screening for 10 rare conditions leads to a 60% referral rate.

If one looks at the risk groups suggested by Mary Sheridan, there are five major groups with 36 subcategories. Table 1 illustrates five subcategories under Family History.

FAMILY HISTORY

Deafness, blindness, neurological diseases, cerebral palsy, epilepsy, etc.

Congenital malformations (including congenital dislocation of the hip).

Mental disorder.

Mother unusually young or elderly.

Family in a "social problem" group.

Table 1

While Dr. Sheridan suggested that her risk list would only select out about 22% of the children, actual experience demonstrated this number to be between 50 and 65% of children. Furthermore, Rogers in discussing the subject pointed out that many handicapped children were not even identified by the risk register. This situation is not surprising if one considers that many handicaps such as hearing loss are 50 times more often acquired in later life than are congenital, and thus are not predictable at time of birth.

4. Failure to Assure that the Identification Methods are Valid or Accurate

Without a full knowledge of the pathogenesis of the specific diseases and handicaps, it is virtually impossible to select and weigh the appropriate risk factors to assure a high degree of accuracy in prediction. Richards and Roberts pointed out that such registers were neither reliable nor were they valid. Reliability or consistency is unobtainable, unless each item is clearly defined. For instance, anoxia, difficult delivery and the five subcategories suggested by Sheridan under Family History are very subjective and very vague. As a result, many subjects may be classified as suspect by one person and not suspect by another. When observations are not reliable or consistent, they are unlikely to be valid or accurate. Richards and Roberts also cite the lack of sensitivity and specificity of the risk register. Sensitivity, as illustrated in Table 2, is the accuracy in detecting all of the diseased or handicapped while specificity is the accuracy in detecting all of the non-diseased subjects.

RELATIONSHIP BETWEEN SCREENING AND DIAGNOSTIC FINDINGS

		Diagnostic Findings		
		+	-	
		a	b	
Screening Test Findings	+	Agreement	Overreferral	a + b
		c	d	
	-	Underreferral	Agreement	c + d
		a + c	b + d	

Sensitivity $= \frac{a}{a + c} \times 100$ Overreferral rate $= \frac{b}{a + b} \times 100$

Specificity $= \frac{d}{b + d} \times 100$ Underreferral rate $= \frac{c}{c + d} \times 100$

Predictive validity of a positive test $= \frac{a}{a + b} \times 100$

Predictive validity of a negative test $= \frac{d}{c + d} \times 100$

Table 2

For example, in an investigation in Aberdeen, few of the 145 mature asphyxiated births showed any obvious intellectual or neurological defects when examined later between 7 1/2 and 11 1/2 years of age. Correspondingly, if only the at-risk children had been examined, a very high proportion of cases of mental retardation would have been missed - 65% if the criterion were abnormal pregnancy and 82% if it were neonatal complications (Richards and Roberts). Thus, the procedures are inaccurate or not valid.

5. Failure to Assure that the Conditions Selected can be Prevented or Ameliorated if Diagnosed and Treated Prior to the Usual Time

As the conditions to be identified were not detailed, it is difficult to determine the degree to which this situation is a problem. However, as yet there are no conclusive studies that demonstrate the efficacy of early treatment of congenital sensori-neural hearing loss and cerebral palsy, two conditions on most, if not all, risk lists. Indeed if early diagnosis and treatment do not improve the outcome, the entire effort is a waste of time.

In summary, the five major problems existing with the risk registers are:

1. Failure to explicitly state what handicapping conditions will be identified.
2. Failure to marshall data that the conditions can be identified at the time of birth.
3. Failure to assure that the problem being sought is sufficiently prevalent to justify the effort.
4. Failure to assure that the identification methods are valid or accurate.
5. Failure to assure that the condition selected can be prevented or at least ameliorated if diagnosed and treated prior to the usual time.

Richards and Roberts summarized the situation in the following statement:

> "The 'at-risk' concept is an unsound basis for the detection of handicapping disorders; there is no alternative to the clinical examination of all infants in the neonatal period, their screening for metabolic and auditory defects at the proper ages, and the careful observation of every infants' developmental progress by doctors, supported by health visitors."

Screening

An alternative approach to early identification of the handicapped is through screening. Screening is the application of quick, simple, economical tests or procedures to an asymptomatic population to single out those individuals who have a high probability of harbouring the disease or problem in question. Thus, screening is not diagnostic. Rather, it is the first step to be followed by diagnosis and, if indicated, treatment. With this definition in mind, it is clear that the use of high risk categories is a type of screening.

Not all conditions lend themselves to screening due to lack of cost efficiency. Over the years, the series of criteria in the selection of conditions for screening has evolved. These are:

1. Serious
2. Possible to Diagnose
3. Prognosis Improved
4. Adequate Lead and Screening Time
5. Treatable
6. Relatively Prevalent
7. Not Harmful
8. Facilities to Diagnose and Treat
9. Cost Reasonable
10. Public Acceptance

Just as there are criteria in the selection of diseases for screening, so there are also criteria in the selection of screening tests. These criteria are:

1. Reliability
2. Validity
3. Appropriateness for Population
4. Acceptability
5. Cost Efficiency

It is my recommendation that one first decide which condition to screen for on the basis of the two sets of criteria. If the prevalence of a disease or handicap varies markedly between two groups, it may be more cost efficient to selectively only screen high prevalence populations. An example is the selective screening for Sickle Cell Disease of Blacks or the screening of Jews for Tay Sachs Disease. As you consider the above criteria you may want to confine your screening to only identify two or three conditions such as problems of vision, hearing and development, since these are prevalent and meet most of the aforementioned criteria.

Comments Regarding the Alberta Program

In regard to the Alberta Health Surveillance program, you have combined universal screening with the At-Risk Concept. In doing so, the following risk concept problems have been avoided:

1. The follow-up of only those subjects at risk.
2. Application of the at-risk method only at one point in time, namely, at birth.

On the other hand, it would appear that a number of problems still need to be dealt with. These are:

1. Failure to explicitly state what handicapping conditions will be identified.

2. Failure to assure that the problem being sought is sufficiently prevalent to justify the effort.
3. Failure to assure that the identification methods are valid.
4. Failure to assure that the conditions selected could be prevented or ameliorated if diagnosed and treated prior to the usual time.

Though the risk approach to early identification of the handicapped has been with us for about 20 years, data is still lacking to demonstrate the extent to which it decreases the number of handicapped children, and therefore improves the health of the population. Perhaps this is a good time for the architects of the Alberta Health Program to evaluate their available data, to determine the efficiency of various aspects of the program and, in light of these findings, to chart their course for the future.

FAMILY RELATIONSHIPS AND INFANT MENTAL HEALTH DISORDERS
SOME ISSUES AND OPPORTUNITIES FOR PREVENTION

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The fields of child development and child mental health have shared an orientation that what is normal or "normative" in development and behaviour is derived from the study of well-nurtured children reared in families that function at least reasonably well. Deviations from those patterns have been considered abnormalities. For the past 100 years at least, the family has been considered the environment in which the child can best thrive. Threats to the family in its nurturing role have been at the forefront of concern in recent years.

Most of us, living in the here and now, have an impression not only of change but of rapid change, if not in family forms at least in the number of families that depart from the nuclear and extended family organizations of the past 100 years or so. One-parent families, headed by mother or father, adoptive families, foster families, communal families and those made up of parts of two previous families, are not new. What is a change is that there are larger numbers of all of these, let us call them for the moment, "untraditional" families, as a result of the choices of adults and of stress and change in the society.

Statistics currently available differ in exact percentages or numbers reported, depending upon the sampling method and the time span covered by the data. However, there is general agreement that there has been a substantial increase in the United States in the past 20 years in the number of two-parent families in which both are employed outside the home; the number of one-parent families whether the result of divorce, separation, death of the spouse or choice of unmarried parenthood; the number of children born to young teenage mothers who are themselves still children; and the numbers of divorces or separations which may cut children off from one or another parent. That these and other threats to family function are a cause for concern among child mental health professionals is reflected in such publications as the series of The International Association of Child Psychiatry and Allied Professions.(1,2,3,4,5)

Solnit(6) has suggested that the family as a social unit in some form must survive because of the needs of children and the adults closest to them. He said ".....the family is not only a nourishing, protective and guiding unit, it is also a meaningful bridge from the past to the future. If we were to decide that the family unit is antiquated or that it had outlived its usefulness, following our best knowledge of human development, we would have to create a form of social environment for children and their closest adult relatives that would have many or most of the features of the family as we know it today and have known it for the past hundred years. Accordingly, it is imperative that we avoid the unnecessary re-discovery of what we know or can predict, and that we find what societal resources can be mobilized to support the family today and as it will evolve in the future..."

Most of us, I suspect, share Solnit's expectation that the family will survive in some form. Most adults will probably continue to wish for and to seek continuity of affection, intimacy and social satisfaction from one another, and to represent themselves in the next generation through having and rearing children. But it has become abundantly clear that parents need the support provided by other adults important to them, and that families need the support of a community that cares about them. Parenthood, too, is a developmental process. The capacity for nurturing the young, for rearing children, starts somewhere and, if the adults are reasonably healthy and life is not too hard, the capacity grows, expands and deepens, as parents meet its challenges, experience its satisfactions, anxieties and disappointments, joys and sorrows. Becoming a parent has been called a normal developmental crisis, calling for a significant adaptive response, and individuals are more or less successful at developing their ability to cope with parenthood at the beginning and over time. Erickson's(7) felicitous words convey much that is significant in this regard: "...a seeming paradox of human life is man's collective power to create his own environment, although each individual is born with a naked vulnerability extending into a prolonged infantile dependence. The weakness of the newborn, however, is truly relative. While far removed from any measure of mastery over the physical world, newborn man is endowed with an appearance and with responses which appeal to the tending adult's tenderness, and make them wish to attend to his needs; which arouse concern in those who are concerned with his well being; and which in making adults care, stimulate their active caretaking... Defenseless as babies are, they have mothers at their command, families to protect the mothers, societies to support the structure of families, and traditions to give a cultural continuity to systems of tending and training".(7) Following Erikson's thought, one can easily recognize situations when child and family are at risk: when the infant has no mother at his command, when the mother does not have adequate protection and help from those closest to her, when the family is placed under great stress because of societal conditions; when the formerly relied upon systems of tending and training become fragmented and confusing or appear to have outlived their usefulness.

What we have learnt, or think we have learnt, about what constitutes good child care, what environments facilitate the child's physical, mental and emotional health, provides some of the guidelines when it becomes our task to develop needed support systems for families. When we are called upon to provide services for individual families, as parents seek help for the infant whose development suffers in some way, this knowledge, comprising the current state of the art, while far from perfect, nevertheless permits reasonable approaches to diagnosis and therapy. We are on the most solid ground when dealing with the individual child and his family, less so when trying to plan large scale programs.

From case studies and systematic clinical research, as Escalona(8) states, *retroactive* study has shown beyond a doubt that particular life stresses, deprivations, frustrations and traumata during the pre-school years are significantly related to psychiatric illness or deviant, impaired development... *retrospectively*, the association between biological risk factors and many environmentally-determined social and psychological risk factors has been demonstrated. At the same time, we know that the same traumatic events that produce maladaptation and illness in those who become patients are also found among large numbers of normal individuals.

Recognition of this fact has led to heightened interest in issues of vulnerability and resilience, of coping, adaptation and mastery. Though there is uncertainty about long range prediction of developmental outcome, there is reasonable agreement about the advantages of recognizing deviations in development and of intervention according to the nature of the problem. This is based on the assumption that facilitating the infant's developmental progress at any one period is likely to better prepare him for the next phase. Conversely, failure to alleviate significant disturbances in functioning may make him more vulnerable to the stresses ahead as well as neglecting what could be done to improve current functioning.

In defining clinical approaches to recognizing deviations and facilitating development in infants and families, there is current emphasis(9) on the importance of viewing human development as involving multiple inter-related lines of development, rather than single unrelated sequences. "Included, for example, are physical and neurological growth, cognitive or intellectual development, the development of human relationships and the capacity to organize and differentiate experience (coping and adaptive capacities)." An intervention strategy which takes into account the existence of multiple lines of development will approach a problem in ways that will facilitate development in all areas of an infant's life. For example, babies who have been nutritionally compromised improve physically and gain weight better when good nutrition is combined with adequate social interaction. A second assumption forming the basis for a clinical approach to preventing and alleviating infant mental health disturbances views the infant in a context that includes not only the multiple developmental lines but his mother, father, other family members and relevant other aspects of the social environment. The approach emphasizes the interaction and interdependence of developmental sequences and environmental influences.

Elsewhere(10,11,12) I have written about characteristics of an environment that facilitates the development of the infant, and reduces the risk of mental health disorders, and I will not go into great detail about it. But in order to establish a frame of reference, I will just list, in condensed form, some of the constructs that I believe are largely non-controversial among most early childhood specialists. The environment that supports the infant's development provides the following:

1. First and foremost is good physical care from a nurturing person or persons who can also provide affectionate care in accordance with the child's developmental needs. The reasons for good physical care are well understood. As to the second, continuity of affectionate care and the formation of close relationships with one or a small number of adults are still considered by many of us to be a necessary pre-condition for encouraging the child's development, and reducing the risk of a psychiatric disturbance.

2. The child needs a speaking social partner. The child's speech and communication in general, and aspects of cognition suffer in the absence of a speaking social partner.

3. He also needs opportunities to act upon the environment, that is, to use emerging skills in a supportive and safe atmosphere. The development of strategies of coping and adaptation in particular is very much influenced by this factor.

4. Consistency and repetition in the child's experience are desirable, especially in the behaviour of his care-givers toward him, so that he finds them predictable and supportive. A great deal of the infant's learning about the outside world and about himself is anchored in the consistency of experience with trustworthy parent figures.

5. Within the atmosphere of consistency and predictability, the young child also benefits from experiences of variety and contrast. These experiences sharpen perception and awareness, creating those mild psychological tensions that call for an adaptive response. When the tension is not so great as to overwhelm and disorganize the child, a novel experience or stimulus can enhance development in a number of ways.

6. The child needs toys and other playthings. While a responsive human partner is crucial to the child's development, he also needs toys that can be used independently and with others. In addition to their usefulness for intellectual growth, toys serve an important function in the child's emotional life. One of their advantages is their emotional neutrality; the child can use them in many ways to work out feelings and develop ideas without evoking an emotional response from the toy.

7. The child needs limits, prohibitions and expectations for conformity from the adults. These are beneficial when provided in an atmosphere of continuity and affectionate attention. Harshness, coldness and severe punishment have no place in rearing a healthy young child, but it is also believed that development cannot proceed in a favourable way for a child whose parents are markedly overindulgent, excessively permissive, or extremely inconsistent in what they require of him. Setting reasonable limits and requirements is as much a part of loving a child and supporting his development as are feeding, cuddling and speaking to him.

8. If he has special needs due to some handicapping condition or unusual sensitivity, these must be specifically addressed as well.

These, then, are some of the general characteristics of an environment supportive of the child's development. We know that these are provided in families that nurture children well, and some version of the above becomes part of the criteria by which we assess the adequacy of parental care.

In addition to what has been learnt from research and practice about environments that facilitate development, there is also some information about the special characteristics of infants that I have found very useful in the diagnosis and treatment of early childhood disorders.

1. The younger the child, the more likely he is to react to adversity with global responses and symptoms, rather than more focussed or differentiated ones. This is a reflection of the closeness of the psychic and somatic systems in infancy.

2. In respect to development, a situation of adversity in the young child's experience such as maternal depression or a chaotic environment where there may be not only deprivation of positive experiences but the presence of negative ones is highly likely to disturb or cause delay in more than one area of development. It also causes trouble in the organization and integration of those functions. An older child who has had reasonably good care and development up to the point of a traumatic event or difficult situation will react, of course, but because of greater maturation, differentiation and integration of functions will have more differentiated reactions, including a more extensive repertory of defenses and coping abilities. An infant of six months may be fearful or anxious, for example, but he will not be phobic; he does not have the amount of psychic structure that permits a phobic projection.

If we agree, as I assume we do, that development is the result of the interaction of biologic and psycho-social factors, then we are compelled to begin any assessment of an infant with a broad view. Moreover, we have to keep in mind:

3. that some infants are more vulnerable than others to psycho-social stress; some not so vulnerable biologically may, nevertheless, be at risk for disorders of development because of adverse experiences. When there is both biological vulnerability and an adverse psycho-social environment, the conditions that produce disturbance in the child are maximized. Prematurity, traumatic birth, primary mental subnormality and sensory defects such as blindness or deafness, when combined with problems in the nurturing environment, for whatever reason, are especially hazardous for the developmental process and for behavioural organization.

I would like to shift now to a consideration of some aspects of your work and my work as professionals providing services for infants, and how quickly considerations of the family environment must be taken into account. I want to start discussing the issues from the viewpoint of the professional who is first confronted with the problem, usually the general practitioner, paediatrician or nurse. We, as health professionals, operate in a social context, and many of the findings relevant for assessing psycho-social development are interactional, i.e., they go on between clinician and child, clinician and parent, parent and child. It is also true that most of the dimensions of treatment of such disorders are interactional also.

I hope that you will forgive that I will give very little time in this presentation to one group of mental health disorders of infancy, namely, the severe neuro-psychiatric disorders of early onset such as childhood autism, atypical personality development and the behavioural and personality problems of infants sometimes associated with primary mental subnormality. I have chosen to focus on those mental health problems in which the child's experience, especially with his care givers, is crucial etiologic factors currently often designated as psycho-social disorders. The choice is based both on the frequency of such problems and on a certain optimism about what can happen if one is there to recognize them and begin to do something about them.

When the infant under three comes into your office or clinic or is seen at home, often you do not know, especially if this is a new or seldom seen child, just what you are going to find. Is he a developmentally normal infant with, for example, an acute infection or a chronic health problem, or an infant with an obvious developmental disability? So, all you will have to be ready to think about, is everything! Sound paediatric knowledge, including a knowledge of child development - what, in general, facilitates or interferes with development - is needed. In that connection, it is known that a paediatric interview and physical examination are of pre-eminent importance in the initial appraisal of developmental problems, including those learned on study to be due to adverse psycho-social factors.

You begin, as usual, by asking the parent to tell you about the child and to some extent about the family. Both parents' ability to provide information and your experience and skill as an interviewer, of course, will be important determinants of whether or not you get the relevant diagnostic data. You will have, in addition to the information from the history and physical examination, the following opportunities:

1. Observations of the behaviour of the infant, both spontaneous and in response to stimulation.

2. Observations of parent-child interaction.

3. Observations of the parents' behaviour with each other if both are there; their attentiveness to the child, attitudes and preferences about child care.

Attention to these three categories - none of which is alien to the paediatric situation - greatly enhances the opportunities for defining mental health disorders of infancy. If one comes into the picture as the psychiatric consultant, much of the work-up may already have been done. Nevertheless, the task, especially during infancy, is not an easy one and, when we consider some of the signs and symptoms, that will be readily apparent. I have elected to be descriptive here, to emphasize what can be discerned in the child's behaviour. Naturally, we are concerned with events within the child's mind also, but we have to depend largely on behavioural cues for our data, and much of that is non-verbal in this age group. The most commonly occurring signs and symptoms of psycho-social disturbances are:

1. Disturbance of physical growth and nutritional status (e.g., failure to thrive, psycho-social dwarfism).

2. Disturbance of sleeping, eating and elimination functions.

3. Disturbances in interactions with others, especially as seen in the parent-child relationship.

4. Developmental delays: for example, delays in motor development, speech, intelligence, social development and in coping behaviour. There is often deviant behaviour as well as delay in the acquisition of skill.

5. Disturbances in affective expression and mood. Among them are apathy, depression, extreme excitability or lability, anger, excessive distressed crying or the lack of it. Problems in modulation of affect are common.

6. Disturbances in the ability to play - both social play and play with toys.

7. To make it more difficult, one must think also of those disorders of infancy that are classified as psycho-physiologic in nature; disorders in which there is a significant interaction between somatic and psychological components. These, as is well known, may be precipitated and perpetuated by psychological or social stimuli of a stressful nature and are especially common in infancy. They may be mild or severe, transient or continuing. Among those seen often in infancy are those expressed as disturbances of gastro-intestinal function, skin and respiratory disorders, neuro-endocrine disorders and the like. They may present as disturbances in state regulation, at first physiological, a little later psychological as well.

One of the characteristics of the work is the need for at least several interviews and observations. I am amazed at our professional megalomania when we expect to know very much on the basis of one contact. It is very important that those who assess the parents include an evaluation of the parent as parent. A psycho-social or psychiatric evaluation of the adult is not sufficient for an understanding of the child's disorder: it is the adult as a parent - and as the parent of a special child patient - that requires assessment and this, I believe, is very difficult to do, if one does not also know a good deal about the child's development, including some of the specifics of developmental needs and what goes into meeting them.

Treatment in regard to these disorders is as it is in medicine in general: there is an implied prediction that what we recommend is likely to help, and we keep a close watch on things, modifying treatment as necessary. The treatment plan will address itself both to the signs and symptoms of disturbance or delay in the infant and toddler, and to the etiologic factors, including the influence of the environmental factors. The fact that this is often easier said than done should not deter us in our efforts to clearly define the diagnostic and treatment issues and methods.

Neither the diagnostic work nor the treatment of so young a child can be successfully carried out without the sanction and participation of emotionally important adults. Acute crises and immediate threats to survival can at times be alleviated with minimal parent participation. But in the face of chronic stress, what has been called strain trauma, including that due most commonly to disturbances in the parent-child relationship - there is no satisfactory substitute for the participation of the adults who will provide the continuing care of the child, whether he is in his own family or elsewhere. The extent to which parents can be helped in their role as nurturers and guiders of the child will be a crucial determinant of outcome, even in those situations in which important additional help in the form of education, substitute care and therapy for the child is also provided.

I suspect that, in principle, no one would disagree with the vital importance of including parents in the treatment plan, but to develop ways of carrying this out is no simple matter. Part of our difficulty in this regard is that, as health professionals, most of us were trained in a tradition that suggests we should arrive at a diagnosis, tell the patient what is wrong and what should be done about it and to expect to follow it through in short order. Mental health professionals know full well that is not so simple. Infant specialists have marvellously difficult lives: patients who cannot talk with them, whose behaviour can only be guessed at. Our dependence on those who know the infant well is tremendous. Older children who are referred or brought by parents to mental health agencies often have already been identified as needing help for psychiatric problems. However, the same cannot be said about children under three years. Since they often present with delayed development or with somatic symptoms as I said earlier, parents are much more likely to be thinking about physical illnesses, mental retardation, brain damage and the like. In such instances, many parents are far from ready to be able to see the part they play in the child's symptom picture.

In a review of 100 consecutive cases of young children under age five years treated at our clinic for non-organic mental health problems, we found, for example, that in 75% there were very substantial problems in the marriage that influenced the child's development adversely.(13) Very few of the couples came to us saying that because their marriage is in trouble the child is suffering. So, in this kind of diagnostic work, there is no substitute for following the old casework principle of starting where the client is. From the beginning, we try in our clinic to create a contact with parents that conveys an attitude that what they know about their child, their concerns, observations and fears are essential aspects of what we need to know in order to arrive at an accurate diagnosis. Similarly, we convey to them that no remedy is likely to be truly successful that does not have their sanction and participation.

When the time comes to recommend a treatment we will have developed at least some kind of working relationship with parents. In so doing we will have arrived at some judgement about how they will receive and react to what we have to say, including their readiness to participate in treatment. We know, also, that even the most concerned parents, who wish to participate, may not be able to modify their behaviour toward the child, because of the nature of their own problems: depressed mothers, for example, cannot just "turn off" their depression, because it would be good for their child; borderline and disorganized parents or those with infantile personalities may continue to have problems in their parental functioning in spite of a wish to the contrary. In the treatment of an infant, then, the close co-ordination of work with the child and work with the parents is essential.

Opportunities for Primary Prevention

In the best of all possible worlds in terms of quality of life, and if we had much more knowledge and wisdom, perhaps we could approach the goal of primary prevention. Since such is not the case I want to restate it in more pragmatic terms: an acceptable goal at present is the earliest possible recognition of risk factors and vulnerability (both biological and experiential), and the mobilization of services that, according to current knowledge, are likely to provide the most effective means of alleviating problems and reducing the possibility of further disturbance and impairment of development.

That is a more modest statement, but it, nevertheless, defines a very formidable task, one that will require the combined efforts not only of clinicians and educators but of the individuals and institutions in the wider society. My focus here will be limited to the work of clinicians and other early childhood and family life specialists.

First, I believe that there is a great deal that can be done with an integrated clinical approach that includes 1) commitment to address, in diagnosis and treatment, the complex issues dictated by the inter-related lines of development that characterize the human child, and 2) an awareness that the infant must be viewed in a context that includes also his parents, other family members and other aspects of his social environment. This is an approach that, as indicated earlier, acknowledges the importance of the interaction and the complex co-ordination of the multiple factors involved in healthy development or its opposite.

Basic to the task of prevention are the familiar methods of the clinician: careful diagnosis and choice of treatment. Accurate and comprehensive clinical diagnosis will be based on knowledge derived from many sources including, to name a few, medical paediatrics, genetics, neurology, psychology and psychiatry, clinical social work and the social sciences and early childhood education. The individual child development specialist will not ordinarily have expertise in all of these fields, and must draw upon the skills and contributions of others. There is nothing new in this statement, of course. The reason I bring it up is that we tend to do it rather badly. Sometimes we overlook or fail to call for the relevant consultation; sometimes our failure to be effective is that we do not co-ordinate and synthesize the diagnostic data from all sources into a coherent diagnostic formulation that leads to a reasonable treatment plan.

In approaching preventive intervention it is useful to consider three large categories of the very young child with mental health disorders:

1. Biologically impaired infants whose handicap is a neuro-psychiatric disorder of early onset - childhood autism is perhaps the best known example (Tourette's is another). These are infants who, in the best of environments, would have moderate to severe problems of personality development and behavioural organization.

2. Biologically vulnerable infants who, in a reasonably supportive and nurturing environment, would not develop mental health disorders. I am speaking here especially of infants with sensory defects, such as deafness and blindness, premature infants, infants with inborn errors of metabolism, varieties of brain damage and many others whose development is at risk on two counts. The first is that the organic handicap, by its very nature, may mean that the infant has special needs, requiring special talents from parents and superior health and educational services. The second is that the birth of a less than perfect child is a cruel blow to parents, and they vary widely in their ability to handle their pain in such a way as to create an environment that optimizes development. In our clinical work, we are usually very aware of those infants in whom the combination of a biological vulnerability and adverse influences in the psycho-social environment result in significant developmental disturbances. One also sees, happily, situations in which parents are able to respond to the special needs of their infants in ways that optimize their development, help them to achieve great things, turning defeat into victory.

3. Biologically normal infants whose disorder is due to the influence of experiential factors, mostly for the very young, problems of adults in meeting their developmental needs, as indicated earlier.

The most effective prevention approaches problems from biological and experiential perspectives and their interaction. Whatever we can do to understand frank biological disorders, and the biological pre-dispositions that influence behavioural organization and the psychological life and health of human beings, needs to be done. This involves everything from basic neurobiological and neurochemical research to the continued collection of carefully studied and treated cases in a service context. It is still true that every case should, by those who provide the service, be considered an opportunity for learning more. I do not mean, of course, that every child should be in a research project. What I do mean is that the observing, inquiring, discerning and compassionate clinician has much to add to our knowledge about how physiology and experience interact, how the developmental progress proceeds, what interferes, what facilitates. I regret that it is currently not very popular to report individual case studies, since there is much still to be learned in our field at least from accumulation of experience and information gained from individual situations carefully documented and followed over time.

On the experiential side, there are a number of important opportunities and challenges of which I will list four:

1. Improving the environments in which we provide services for children. Sometimes this is called "humanizing" them, because we have had many facilities - especially in health care and in those settings that provide substitute care for infants without families - that are clearly recognized as inhumane and damaging, though usually run by persons whose goals are laudable ones. Perhaps we will avoid some unnecessary acrimony, if we speak of providing environments that take, as their guidelines, the health and developmental needs of infants and work more carefully at how to plan these environments and how to make them work more effectively. There are good models for such care as well as bad ones; good programs as well as suspect or noxious ones.

2. Improving the preparation of those who provide the services to deal more comprehensively with the whole child and the adults with whom he lives. The current deficits in training are appalling, especially in regard to details of child development and the importance of psycho-social issues in the well-being of the young child.

3. Co-ordinating professional efforts. Because the necessary skills are not encompassed by any one discipline or area of training, it is essential that much more effective methods coordinate the services and

skills, especially of health, mental health, social and educational services.

4. Integrating services. In regard to infant mental health, I do not believe we will ever have truly effective early recognition and preventive intervention, until mental health professionals, or the knowledge and skills they have, are an integral part of the primary health care system. It is easier to include them in tertiary care centres or to use them as resources for referral, and this is important too. Such resources will always be needed. But the factor of time, in the life of the very young and the vital importance of timely treatment, makes it essential that we do something more about providing help in the paediatric context. The fact that there are, in most communities, not enough services, not enough persons skilled in the earliest years of life, is regrettable, but it is also true that our current ways of working do not make best use of the talent we have.

In conclusion, there is much work to be done if we are to be successful in preventing disabling mental health problems in young children. The challenge is very great and one that must be addressed by all of our societal institutions - not by clinicians and educators alone. What we can do as professionals serving children and families, and this is no small achievement, is to apply the knowledge we have toward the early recognition and alleviation of risk situations and special vulnerabilities and this is more than enough to keep us busy for some time to come!

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MATERNITY/NEWBORN CARE

PSYCHO-SOCIAL CARE IS GOOD PREVENTION

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Introduction

This paper will attempt to acquaint you with the present situation and to present some of the research that indicates changes are needed. We make a number of recommendations that we ask you to seriously consider in your hospital and community.

During the past 10 years, there has been an increasing awareness that psychological and social factors play an important role in maternal-newborn care. Everyone seems to agree that changes are needed to make childbirth a joyous experience for families while reducing perinatal mortality and morbidity. Everyone wants to provide the healthiest babies possible, but everyone does not agree on how best to do that. Attitudes, policies and procedures surrounding maternity and newborn care vary widely from hospital to hospital, family to family and province to province, as indicated in a recent survey of 392 Canadian hospitals by the C.I.C.H.(1)

Childbirth today is generally perceived as a medically safe event in a woman's life. Hence, while, traditionally, the pregnant woman accepted "current" medical practices for her baby without much questioning, today's child-bearing women want to maximize this experience for both themselves and their families. Most lay consumers are aware of the concept of maternal-infant bonding and how this bond can be facilitated by a close maternal-paternal-infant association around the time of birth. Even mothers of premature infants no longer accept off-limits rules and fight to see and touch their babies.

Some hospitals and doctors are responding to this increased consumer demand for more individualized and natural childbirth experience by insisting on fewer "routines" and "policies" during the actual birth process. A few hospitals have gone even further and developed a "family centred obstetrical service". Such a service is based on a philosophy that allows parents to make decisions about which childbirth experience is best for them. Along with the parents' rights to make these decisions, comes the responsibility for them to be aware of the implications of the alternatives they choose, and the responsibility to be educated in labour breathing techniques, breast feeding and baby-care in order to assume these roles themselves in the family birth experience.

The family also has the responsibility to discuss their needs in advance with their doctor, and to obtain clearance from the hospital where they will deliver.

In 1977, George Engel(2,3) presented an alternative model of medical care, and called it the Biopsychosocial Model. This Model sees an "illness" to be a disturbance in a complex integrated system. A particular illness is, at the same time, a component of higher systems, just as a person is a component of a family or society at large, as well as of lower systems. Engel states that the system's oriented clinician will be aware that his primary task is the identification and characterization of the abnormal constituent components of the system (i.e., the illness) he has to deal with. This knowledge will then lead him to intervene at the system(s) level most appropriate for the disorder at hand, but always allow him to remain conscious of changes within other systems which may affect the future process of the disorder. Let me give you an example where I believe we are beginning to do it: cancer - deal with shock - reintegration into school - holistic medicine - children - medication - bald head - other children.

For a pregnant woman this implies that the biopsychosocially knowledgeable physician or other health care professional tries to understand the effect and possible repercussions of her present condition or pregnancy on her total life space, and attempts to meet her various needs as they arise. It is obvious that at present only a minority of health care professionals practise this type of medicine.

In practice, many hospitals in Canada(1) retain certain "routines" and "policies" which, by their very nature, interfere with a biopsychosocial approach to childbirth of low- as well as high-risk infants. Some of these include: during labour and delivery, the routine shave prep of perineal hair, an enema, analgesia and anesthesia, delivery in a "delivery room" which resembles an operating room, delivery in stirrups in lithotomy position on a delivery table, almost routine episiotomy, silver nitrate administration to the newborn at delivery, and separation of mother and infant after birth for a period of up to 12 hours.

During the postpartum period, these routines include: restricted rooming-in policies which exclude overnight stay of the infant, routine feeding times even for breast-fed infants, routine supplementing of breast-fed babies with glucose and water or formula from a bottle, restricted family contact with newborn, lack of sibling visiting and contact with the new baby and lack of grandparent contact with the new baby. In case of the delivery of an ill or premature infant, such "routines" often include: restricted visiting of the infant in the high-risk nursery, restricted contact with the baby, delayed breast feeding replaced by bottle feedings, transfer of a sick infant to a specialized neonatal unit (frequently without any provision for the mother to accompany her infant) and lack of explanation of procedures and plan of care for the infant to the mother and/or her family. (See appendix.)

All of these "procedures" tend to disrupt the natural process of childbirth unnecessarily, and by causing anxieties with the new parents, unduly compromise the positive beginning of the parenting process.(4)

In a family-centred system, the role of the physician and hospital is one of supporting the couple to have the childbirth experience of their choice, to be available when complications arise and always to communicate with the parents.(5) Family-centred care provides the most clearly defined challenge to the traditional form of obstetrical practice for uncomplicated pregnancies and the low-risk infant. Every child has the right to be well and safely born, but this does not contravene the right to be born in a nurturing and emotionally supportive environment. Family-centred care consists of an attitude rather than a protocol. It recognizes birth as a vital life event, rather than a medical procedure. This is well expressed in the Bulletin of the Society of Obstetricians and Gynaecologists of Canada.(6)

> Family centred reproductive care cannot be achieved by merely altering the physical environment or changing the specific policies and procedures that accompany birth. It requires a more fundamental change in approach, with acceptance and understanding of a new, mutually responsive relationship between care giver and care receiver.

Psychological factors at the time of birth and in the early days of life have also been found to contribute to pregnancy outcome. Some maternal conditions (i.e., teen age, substance abuse, maternal emotional illness and mental retardation) will also render the early parenting process more difficult, and substantially increase all types of parenting disorders.(7) In addition, however, there are a number of other conditions which can jeopardize the early parent-child relationship and weaken later parental competence. It is recognized that all parents of premature or sick infants receiving care in a tertiary neonatal intensive care unit undergo profound emotional stress and deprivation.(8,9,10) It is also generally acknowledged that the transfer of a premature or sick infant away from the mother's hospital to a tertiary care unit increases the anguish and anxieties normally associated with the birth of such a high risk infant(11) - although early suggestions that such a procedure would permanently hinder the establishment of a secure, affectional and emotional bond between mother and infant have been shown to be exaggerated.(12)

The literature also suggests that prematurely born infants are more frequently physically abused by their parents than are full-term infants.(13,14) However, a careful analysis of the papers on which this assumption is based reveals that a substantial number (33% in Elmer and Gregg's group, 50% in Klein and Stern's group) of the prematurely born abused infants had also been mentally retarded prior to the actual abuse. This means that premature infants who suffer postnatal complications are in an especially high-risk group. The reasons for these findings are two-fold.

Premature infants, even when they have reached the age of their expected date of birth, show an inferior ability to respond to outside stimulation than do full-term infants.(15)

It has also been found that severely ill premature infants show significantly less activity during random observations in the neonatal intensive care nursery than do comparatively well premature infants. More importantly, however, their mothers also interact with them significantly less than do parents of comparatively well infants, matched for gestational age and maternal visit number. This relative lack of interaction continues at least during the first three months at home, long after the infants have recovered from their neonatal complications.(16) More significantly, the mothers of infants who were severely ill for less than 17 days appeared able to adjust their own behaviour when their infants had recovered medically, while those whose babies were critically ill for more than 30 days never responded behaviourally to the recovered state of their infants. This may indicate that a very long and serious illness during early infancy can so affect the primary caretakers that they will fail to perceive, at a later time, their infant as he is in reality, but continue to behave towards him according to an inner perception set up during initial illness.

It is clear from the foregoing that a good perinatal care system should:

A. Provide every pregnant woman and her family with the maximum of social and psychological support during her pregnancy and her initial period of parenting.

B. Identify early those families who represent a psycho-social risk as far as their pregnancy and/or parenting style is concerned.

C. Treat and/or refer for assistance those cases who require help not available within the routine perinatal care system.

D. Develop a co-ordinating body which can monitor governmental and hospital policies, as well as medical practices that clearly violate the premises of points A to C.

After careful consideration, Dr. Minde and I would like to make the following recommendations for a system of psycho-social care for families and their newborns.

A. Prenatal Care

1. Every woman who comes for her first prenatal visit to her obstetrician or general practitioner in addition to a routine medical examination should have at least one interview during which her obstetrician or a designated assistant has the opportunity to take an adequate social history, and which also allows the physician to establish a possible rapport with the future mother. Such a procedure would also permit the physician to identify women who: e.g., have no husband, lack an adequate family support system, have had troubles with other children previously and had themselves lived away from their parents for more than three months before the age of ten.

2. Every woman should be referred, during the first trimester, together with any other family member the mother wants to be involved, for an initial series of pregnancy classes which provide information on the importance of nutrition and other substances on the infant and the psychological changes and feelings associated with pregnancy. This instruction may be provided by public health nurses or childbirth educators or midwives associated with an obstetrician.(17)

B. Intrapartum and In-Hospital Antenatal Care

The hospital should provide or see that:

1. Educational programs exist for its medical, nursing and administrative staff which increase their attitude of flexibility and support for alternatives in childbirth, if they are medically indicated. Labour and delivery nurses must be prepared to better understand and support parents in labour and delivery.

2. Husbands have the free choice to remain with their wives during labour and delivery. This is also applied to routinely permitting a prospective mother to have one person of her choice with her during her labour and delivery, if the husband is not available. Such a practice has been shown to significantly decrease neonatal complications and the emotional stresses of the mother.(18) For the same reason, care should also be taken to avoid leaving mothers alone for a long period in small, confined labour rooms.

3. There is no routine prep, enema, intravenous, monitoring, or drugs, unless based on individual situations and joint decision between doctor and patient.

4. Delivery is in the labour room, barring complications with delivery and position of choice, i.e., no routine stirrups or lithotomy position.

5. There is no routine anaesthesia.

6. There is daily contact between mother and infant.

7. First breast feeding occurs immediately after birth, if so desired by mother. There should be improved assistance with breast feeding in hospital by permitting experienced La Leche League mothers to visit the postpartum floor, and teaching new mothers about their feeding.

8. Mothers, fathers and siblings are routinely given the choice to see and touch their infants soon after birth, even if these infants are sick and require special paediatric care.

9. There is no separation of mother and infant, if so desired by the mother, i.e., total rooming-in. Demand feeding even if child is placed in a nursery.

10. There are open visiting hours for husband and siblings.

11. Early discharge for mother and baby (between 18 and 36 hours) is facilitated when support by public health and home care team is available.

12. Following discharge of mother from hospital, a follow-up contact to check on the infant's problems and progress is available.

13. In addition, hospitals are encouraged to facilitate the development of one or more "birthing rooms" with home-like atmosphere, where labour and delivery can take place without the presence of machines and medical equipment.

14. Finally, that professionally prepared obstetrical nurses (midwives) are available. These persons could eventually provide much of the needed biopsychosocial care for mothers and their families during the crucial pre- and post-natal periods.(19)

C. Postnatal Period

1. In order to assist the parents of all infants in their caretaking tasks, and to provide the milieu within which families of high psycho-social risk can be recognized and effectively assisted, each nursery or the neonatal unit should have a limit of 20 beds. Large units are unwieldy (medical staff do not know the nurses, parents see new faces each time they visit, etc.) so that any psycho-social support system is doomed to fail.

2. Flexible visiting and calling hours for all those adults and children of the infants' families whom the parents consider important should be routinely available.

3. The physician who provides care to the infant should be identified to the parents, and he/she should actively approach the parents and tell them of the infant's progress.

4. Nurses with special education and skills in the care of mothers and babies should be sought for the obstetrical, nursery and neonatal units. These nurses should also have an understanding of the attitudes, feelings and grief reactions of parents to critically ill newborns, and be able to recognize parents responding in an inappropriate manner who need to be referred for counselling.

5. There should be sufficient space for parents to visit comfortably. A rocking chair or comfortable seating should be available. Facilities should also be available for breast feeding and for rooming in, where necessary.

6. Each unit requires access to a social worker. This worker should have special skills in the work with high-risk parents and with grieving parents, and be actively consulted by the physician in charge of the infant about any disposition and future medical plan affecting the infant and his family. The social worker should also ideally have access to a child or family psychiatrist who has experience with difficulties encountered in this area of medicine.

7. Every intensive care nursery also requires a community nurse, who will co-ordinate the psychological care of each family by, e.g., visiting each family at home, obtaining a basic family history and assessing the special needs of the particular families. In addition, she may, together with the physician, make referrals to the social worker of the unit of families who face obstacles above and beyond normal parental grieving process. She may also be involved in the organization and running of parent groups.

8. There should be a parent support group to assist members in dealing with emotional stress due to hospitalization of their sick newborn baby in the intensive care unit. A parent hour, held weekly, and conducted by a special nurse in conjunction with a "veteran" parent (i.e., a parent who has had a baby in such a unit during the past year), can be very helpful. In smaller units, parents with a sick

infant may be individually linked up with a "veteran" family so as to obtain both emotional support and guidance as to the special needs of premature and/or sick infants, and in this way provide support comparable to that of an external family. Such a program has recently been evaluated and found to be of both short- and long-term benefit to the infant and his family.(20,21)

9. Ideally, each mother should have the option to sleep for one or two nights in the hospital in one room with her baby prior to her infant's discharge. This will allow her to overcome her usually present fears of being an inadequate mother in a possible emergency, and, together with her previous positive experiences with the ward personnel, will make her much more prone to attend later follow-up visits and carry out the physician's orders.

10. A final recommendation is made regarding U.I.C. benefits. At present, any woman who delivers an infant gets 17 weeks pregnancy leave, of which 15 are paid. The leave can be taken any time from 36 weeks gestation onward, but must be begun following the infant's birth. This implies that a woman who delivered a baby after 26 weeks would have to return to work 17 weeks later, i.e., when her infant is 43 weeks post-conception. In reality, this may mean that she has to go back to work just when the infant comes home from hospital. It is recommended that the U.I.C. benefits could be drawn at any time from the premature infant's birth to his 40th post-conceptual week. This would allow some mothers to continue working until their infant comes home and draw their benefits for 15 weeks from then onward. A nearly identical recommendation has recently been made by the Senate Standing Committee.(22)

Finally, are consumers really aware of the need for change, for more attention being placed on psycho-social care? In a 1980 survey of 295 new mothers from Peel County done by their regional health council, nearly one quarter of the mothers wanted some improvement in the care they received in hospital. Nearly one third wanted improvement in the availability of general information on pregnancy. These mothers also strongly approved of hospital policies that maintained family involvement and parent-infant contact.(23)

In Britain in 1979, when a similar survey was done, women rated hospitals as "exceptionally good" where they felt "freedom of choice and respect for the individual is linked with human caring; where one's partner is given a warm welcome, there is opportunity for full discussion concerning medical procedures, support for coping in labour in whatever way one wishes and where it is accepted that the baby belongs to the parents and is not the property of the hospital."(24)

Individual women have differing needs and expectations of care during pregnancy and childbirth. Sometimes these wishes of individual women will conflict with those of individual health professionals or with institutional policies. It is essential that these conflicts be resolved in a way that provides minimal physical risk for both mother and baby and in an atmosphere that is supportive of the family unit. There is no reason why professionals and hospitals of all sizes and in every province cannot make an effort to adopt practices that are family-centred, and meet the individual needs of that family unit. Psycho-social care during the reproductive and newborn cycle would be good preventive medicine for approximately 360,000 Canadian families giving birth every year. This is the kind of care I would like for my daughters and my grandchildren.

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This paper has been prepared in collaboration with Dr. Klaus Minde. He and I had served, in 1980-81, as members of the Reproductive Care Advisory Committee to the Ontario Minister of Health. Dr. Minde is a child psychiatrist who has carried out extensive research involving seriously ill neonates and their families. We have both come to feel that psycho-social support for families during the reproductive cycle is crucial to the well-being of parents and their babies, not only at the time of the birth, but to their future health and well-being.

APPENDIX
POLICIES AND PROCEDURES BY PROVINCE - ALL HOSPITALS

Policy or Procedure	B.C.	Alta.	Sask.	Man.	Ont.	Que.	N.B.	N.S.	P.E.I.	Nfld.	N.W.T.	Yukon	All Units
May ambulate during labour	56 (100%)	49 (100%)	26 (96%)	28 (100%)	131 (100%)	50 (100%)	15 (94%)	19 (100%)	4 (100%)	9 (100%)	2 (100%)	-	99%
Perineum routinely shaved	29 (52%)	33 (67%)	14 (52%)	19 (68%)	77 (59%)	35 (70%)	14 (88%)	13 (68%)	3	9 (100%)	-	-	63%
IV's routinely started	11 (20%)	7 (14%)	4 (15%)	10 (35%)	35 (27%)	41 (82%)	5 (31%)	2 (11%)	-	2 (22%)	-	-	31%
Food/Drink available to unmedicated women	47 (84%)	40 (82%)	22 (82%)	16 (57%)	100 (76%)	33 (66%)	12 (75%)	17 (90%)	2	7 (78%)	2	-	76%
Analgesia/Anaesthesia to most patients	22 (39%)	26 (53%)	12 (44%)	13 (46%)	91 (70%)	33 (66%)	11 (69%)	10 (53%)	3	6 (67%)	1	-	58%
Choice of delivery positions available	35 (63%)	21 (43%)	13 (48%)	17 (61%)	58 (44%)	16 (67%)	5 (31%)	10 (53%)	3	1 (11%)	-	-	48%
Lights kept low	21 (38%)	15 (31%)	7 (26%)	8 (29%)	35 (27%)	35 (70%)	6 (38%)	9 (47%)	1	4 (44%)	1	-	36%
Routine episiotomy for primiparas	10 (18%)	23 (47%)	13 (48%)	11 (39%)	47 (36%)	27 (54%)	7 (44%)	9 (47%)	3	6 (67%)	1	-	40%
Routine fetal monitoring of uncomplicated labour	17 (30%)	24 (49%)	14 (52%)	12 (43%)	34 (26%)	45 (90%)	12 (75%)	13 (61%)	3	4 (44%)	1	-	45%
Special mirrors for mother to view birth	50 (89%)	41 (84%)	17 (63%)	19 (68%)	114 (87%)	50 (100%)	11 (69%)	8 (42%)	1	3 (33%)	2	-	81%
Infants routinely given a warm bath	3 (5%)	1 (2%)	-	-	8 (6%)	9 (13%)	-	-	-	-	-	-	5%
Father may handle infant in Delivery Room	51 (91%)	44 (90%)	26 (96%)	20 (71%)	120 (92%)	49 (98%)	13 (81%)	16 (84%)	4	7 (78%)	-	-	90%
Infant to breast in delivery area	55 (98%)	45 (92%)	19 (70%)	19 (68%)	102 (98%)	42 (84%)	11 (69%)	16 (84%)	4	5 (56%)	2	-	82%
Infant with mother for one hour after birth	49 (88%)	33 (67%)	18 (68%)	16 (57%)	97 (74%)	33 (66%)	7 (44%)	15 (78%)	3	2 (22%)	1	-	70%
Total	56	49	27	28	131	50	16	19	4	9	2	-	

MS167H

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HS170H

HEALTHY BABIES - EVERYONE'S BUSINESS

THE MANITOBA COMMUNITY TASK FORCE ON MATERNAL AND CHILD HEALTH

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Community Task Force on Maternal and Child Health
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In this symposium, we have learnt about attempts to measure and prevent handicapping conditions of prenatal and perinatal origin. However, as concerned health care providers and planners we must address ourselves to the important problem of how this medical and social information is translated to those that could benefit most - the pregnant woman and her family.

New medical techniques and diagnostic tests now make childbirth even more complicated for women and their families. It becomes increasingly apparent that it is necessary for the community to become involved in the planning of services for the pregnant mother and her baby. With medical costs skyrocketing, and most provincial governments admitting that lifetime medical and custodial care of each handicapped person costs $1 million, the prevention of unnecessary handicap would seem a most desirable goal for community planners as well as all levels of government.

How did Manitoba's volunteer Community Task Force, of which I am the Chairman, originate?

Formation of the Community Task Force on Maternal and Child Health

In January 1977, the Social Planning Council of Winnipeg adopted as a policy priority, "the development of an understanding of and possible model for a preventive, comprehensive, developmental approach to service planning and delivery". The concept of prevention was to be made the highest of priorities and Social Planning Council researchers, after an extensive search, wrote a position paper called "PRESCRIPTION FOR PREVENTION".

In this position paper, four major topics were addressed:

1. Concepts and definition
2. Critical review of current models of services
3. The Human Services Model - Choice for Prevention
4. Evaluation of Preventive Services

Because the Social Planning Council Board of Directors was anxious to alert public and private decision-makers to the importance of prevention, a volunteer Task Force was formed to look at that most crucial intervention point - the mother's pregnancy and her baby's first year of life. Firstly, on humanitarian grounds and, secondly, since handicap originating in early life results in the highest eventual cost to the community compared to any other disabling event, the goals of the Task Force were to look at the prevention of unnecessary death and handicap amongst pregnant women and their infants.

Emphasis was to be placed on suggesting practical improvements to existing services. Hopefully, such improvements should provide financial benefits to our over-burdened health care system.

Task Force Membership

When Manitoba's Community Task Force began in March of 1979, a three-year plan of action was designed.

Each of the 50 volunteers, who represented the University Departments of Obstetrics, Paediatrics, Social and Preventive Medicine, Nursing, Law and Social Work as well as hospital and public health nursing, teachers, psychologists and consumers of obstetrical services, joined one of the seven Task Force Committees. Before designing a model for change, it was essential that the Task Force knew what was already in place. It soon became clear that many Manitoba professionals were trying very hard to provide good maternal care, but the linkages between support systems were tenuous and no one government agency provided an umbrella to oversee maternal and child health services in Manitoba.

Task Force Structure

To develop community awareness of changing needs and to select priority issues, community representatives must be involved in the planning process. Our seven community committees set their own priorities, and recommended prevention strategies that included demonstration projects, education and social action. These committees were:

1. Pre-Natal Services
2. Obstetric and Newborn Services
3. Early Infant and Family Support Services
4. Maternal and Child Health Statutes and Regulations from Canada and Other Jurisdictions
5. High Risk Groups
6. Maternal and Infant Nutrition
7. Public Education

The Task Force has felt that our attempts to develop community awareness through public and professional seminars, and that the selecting of priority issues in prenatal and postpartum care in Manitoba was essential before a community will become interested enough to attempt the reorganization of perinatal services.

Seminars have been presented to health care professionals, and a Speakers' Bureau provides Task Force members who will speak on maternal/child health issues to community groups. An extensive T.V. and radio campaign, targeted at the high risk pregnant woman, was directed by the Public Education Committee in October 1981, and was funded by the Health Promotion Directorate.

In the next two months, we will be publishing papers on Adolescent Pregnancy in Manitoba: Current Status, Future Alternatives, and on the Manitoba Native Indian Mother and Child: Discussion Paper on a High Risk Population.

A Plan for Maternal and Child Health in Manitoba

This major paper will serve as our final report. It should be noted that after two and a half years we already see positive results being generated as existing programs are being changed, suggesting that any difficulties experienced and initiatives on the part of the Task Force have been well worth our efforts.

Some of the Task Force's many concerns have been disparities in health status and the rise of health services, lack of preventive services, late initiation of pre-natal care and the issues of manpower and training.

A main recommendation of the Plan is that the Government of Manitoba establish a Maternal and Child Health Directorate. The Directorate would co-ordinate and augment services in the area of community and personal health services, support services for families and at risk mothers, as well make an improvement in the maternal and child health statistical and record keeping system. Services to Manitoba's pregnant women and their children are now dealt with by four separate government departments.

The loss of citizens in the perinatal period and during the first year of life is higher than the rate in the next 54 years(1), and this area of health is now viewed as the highest priority, as reflected by the decision of the Winnipeg Social Planning Council, the Government of Manitoba and other community funders to assist in the forming of a citizen's Task Force to look at the prevention of handicaps of perinatal origin in Manitoba.

The Task Force is delighted with the extent of community involvement in a prevention study of this size and scope. The complexities of a 50-member volunteer community task force which includes not only sub-committee meetings, but keeping members informed and up-to-date has been well worth the effort. The community has made liaisons with the medical profession, and in return the health care professionals have had a chance to respond to community concerns.

We do not yet know to what extent our Provincial Minister of Health will implement the plans of the Task Force, but to focus the public's attention on the important issues that the Task Force has researched and raised, a conference will be held in June 1982 to discuss the implementation of our Plan for Maternal and Child Health in Manitoba at which time we will make our final report to the citizens of Manitoba.

Unfortunately, the gestation period of reports is less precise and often longer than that of babies. Only time will tell how successful our Task Force has been in making Manitoba the safest possible place for women to have their babies.

RESOLUTION - INSTITUTION OF PRE-NATAL FAMILY ALLOWANCES IN CANADA

RECOMMENDATION

The Community Task Force on Maternal and Child Health recommends that:

> The Canadian Public Health Association endorse, publicize and take positive action to bring about legislative change in regard to the following resolution.

WHEREAS the nine months "in utero" are as important in the development of a child as any nine-month period in the life of a child; and

WHEREAS early identification of pregnancy and subsequent regular pre-natal care have been demonstrated to improve the outcome of pregnancy; and

WHEREAS one of the most important developmental factors which determine the outcome of human pregnancy is maternal-fetal nutrition; and

WHEREAS early contact with the health care system will facilitate access to prenatal education and risk identification; and

WHEREAS early and increased pre-natal education will result in self-reliance on the part of the parents; and

WHEREAS incentive programs in other jurisdictions have demonstrated the use of service and early identification of high risk; and

WHEREAS further economic costs are not desirable, the Family Allowance should begin pre-natally, and continue until the age of 17; and

WHEREAS there is no need for further system service costs because the Family Allowance program is currently in place.

BE IT RESOLVED THAT

The Government of Canada after consultation with the Provinces and Territories:

a) pay a pre-natal child allowance to a pregnant woman, as soon as possible after the confirmation of a pregnancy and the decision not to terminate it, in order that the woman have the financial resources to feed and care for herself properly during the pregnancy as is essential for the future physical and mental health of the unborn child; and

b) pay this allowance on the condition that the pregnant woman receive continuing nutritional guidance and medical care as recommended by a physician or a public health nurse.

Resolution adapted from: Ministry of Supply and Services Canada, Child at Risk: A Report of the Standing Senate Committee on Health, Welfare and Sciences. Ottawa: Supply and Services Canada, 1980, pp. 17-18 3(a), (b).

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HS167K

THE LAW AND REPRODUCTIVE CARE

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Community Task Force for Maternal and
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The Community Task Force on Maternal and Child Health was set up by the Social Planning Council of Winnipeg with the co-operation and endorsement of the provincial Department of Health and Community Services, in 1979, as a result of a study on the prevention of handicapping conditions. The study revealed a relatively high infant morbidity and mortality rate in Manitoba. The Task Force was designed to operate for a three-year period with a mandate to address itself to the problems of unnecessary death, disease and handicap among pregnant women and infants. The emphasis is to augment, or enrich, existing services, resulting in financial benefits to the health care system. The terms of reference provided that the Task Force would look at the year before and the year after birth. When I was asked to join the Task Force I had particular interest in the area due to the fact that I was in my first trimester with my first child.

The Task Force evolved to be approximately 55 professionals and consumers involved in the delivery of services to or concerned with the prospective mother and newborn child. Originally, I was the only lawyer amongst a group of health professionals. As a result of my uniqueness, rather than qualifications, I was appointed to the Executive of the Task Force and assigned the duty of chairing the Committee on Law and Reproductive Care. The seven other committees as the Task Force in the first year were geared towards an analysis of health care delivery systems, and my committee was to provide a resource of information on how identified objectives might be met through the use of legislation.

It became obvious very early that we were not familiar with the law in this area, in spite of the expertise of my committee, including the Executive Director of the primary child care agency in the Province of Manitoba. We therefore expanded the committee to include two other lawyers. After an initial attempt to identify the law in our jurisdiction we recognized the need for a more concentrated effort, not available from people employed full time.

As a result of our identified need, the Health Promotion Directorate of Health and Welfare Canada provided us with funding to employ two law students through the Summer Student Employment Program who prepared, under the supervision of the committee, twelve working papers in the area of the law and child bearing families.

These papers are available as a result of the Health Promotion Directorate seeing fit to pay for the publication of 500 copies of each, which were distributed in the Spring of 1981. The areas we looked at arose primarily from the discussions of the Task Force's areas of concern. We had identified the work place, ignorance of medical history in adoption and the effect of bonding in adoption as areas where we might prevent handicapping conditions. As a result, we produced papers on Adoption and the Acquisition and Disclosure of Records, the Issue of Bonding and the Adoption of newborn infants, as well as The Law and the Illegitimate Child. In the work place legislation we looked at employment standards through the Employment Policy and Practice paper as well as impact of family income during pregnancy and infancy through the Family Income Maintenance and Tax System paper and the paper on the Legal Regulation of Mutagens and Teratogens in the work place. In terms of the health care delivery system, which impacts on the mother and child particularly, we looked at Midwifery Law and Pregnancy Termination from the point of view of Canadian law and general practice and The Rights of the Fetus and Rights of the Father.

In order to identify where the system might be changed or where the control lay with respect to affecting change, we looked at the Funding Arrangements of the Social Services and the operation of the Health Care System from a legal perspective.

The focus of the Task Force's work is obviously the best outcome possible of the first year of life. The law in this area was identified as a potential tool to facilitate this outcome. Problems arose, however, when we tried to identify areas for change. In almost every instance, there were competing interests that made our recommendations difficult.

For example, it was clearly identified that adopted children have a much higher incidence of learning disability and need for psychiatric services than do non-adopted children. It was suggested by research in the area that this may be traced to a gap in bonding and attachment during the "placement" process, particularly the first 30 days of life.

Agencies responsible for placement strongly encourage the natural mother to nurture the infant in its first few days of life and then separate before making final decisions on surrendering for adoption. Their experience suggests that this is much healthier for the mother as it permits a "mourning" process. They, therefore, support the legislative restriction on signing authorization for placement documents to no earlier than the ninth day of life. Depending on weekends and holiday schedules infants may wait with no primary caretaker after initial bonding has started for three weeks until placement in the adoptive home.

The research is clear that no matter how strong the relationship in the adoptive home is, the deficit in bonding can never be made up. The jurisdictions such as Alberta, without a mandatory waiting period, report that their experience on immediate placement leads to no more than 3% of mothers changing their minds. Our paper, therefore, reviewed the different provisions in law and the corresponding experience.

Genetic counselling toward the prevention of handicaps leads us to look at the rights of illegitimate and adoptive children to access to medical histories. Education about the value and necessity of a genetic/medical history seemed of prime importance, but, with the help of the Manitoba Law Reform Commission, we saw the passage of a Central Registry for medical and social histories of adopted children and their biological parents, which can be accessed for this purpose. The anonymity is to be maintained through the use of non-identifying data.

A major concern identified was the late identification of pregnancy by mothers. This often in young women was denial, in working women was because of the threat of job security and loss of income security, and in women from remote areas was due to an unwillingness to be removed from home for the birthing process. The net result was a large number of unnecessary high risk situations. In terms of remedies, therefore, we looked at a number of alternatives. It was clear that a number of women might identify earlier, if there was some incentive. Complacency since the last pregnancy had turned out well might need a relatively small incentive. We drew on the European experience in France and Finland where small tokens and positive reinforcement contributed to the turn around in their mortality statistics.

We identified the possibility of an early commencement of the family allowance upon medical certification of pregnancy as a relatively easy and universal option. We met with Monique Begin, at her request, as a result of our initial approach. She identified the Task Force as advocates for the babies who cannot vote and has continued to respond to information sent on other aspects of the Task Force's work.

We looked at prenatal family allowance from two perspectives, the law, i.e., the legislation, and the economic considerations, i.e., relative cost. We identified that if 40 multiple handicapping conditions could be identified early and prevented in Canada each year, the plan would more than pay for itself.

We proposed payments to be suspended if confirmation of the condition of pregnancy or birth was not filed after three months of payments commencing. The administrative adjustments are relatively minor. The result of our paper has been a major recommendation from the Task Force for early commencement of family allowance for which we are seeking national support.

The pressures on working women, we addressed with our paper on maternity/ paternity leave provisions across the country, employment standards provisions and some collective agreements and public/civil service statutory protection. We looked at U.I.C. benefits structure, and recent demands for paid maternity leave. We looked at income security mechanisms already in place through tax exemptions and tax credits and refundable tax credits.

Finally, we looked at the regulation of Mutagens and Teratogens in the work place. This area was particularly fraught with competing interests. Full knowledge of the impact of Mutagens would discourage men as well as women from working in many work sites. The threat of Teratogens would discourage hiring of any women. The impossible short-term solution is, of course, a clean work place.

We reviewed the limited jurisprudence in the area of putative fathers and rights of the unborn child as possible vehicles for bringing attention to the need for improvement in our mortality and morbidity statistics.

All the papers received national distribution, and have received very positive comments. We hope that they will continue to be useful in the evolutionary process of improving outcomes for new babies and mothers. Generally I would say that this whole area suffers from a lack of jurisprudence, but not a lack of regulation. In other words, there is a great deal of law without very much examination of that law before any legal tribunal.

For the purpose of our committee's work to go further, we had to identify what our optimum goal would be towards the prevention of handicap. We identified, for example, that our prospective mother should leave the work site at least two months prior to delivery and not return before six months after birth. On that premise, we will look at what recommendations for change are achievable. We have met with our Attorney General who has indicated his willingness to make some changes and to refer some identified areas to the Law Reform Commission.

I would be pleased to discuss the specifics in any area you would like expanded.

HS163D

ASSESSMENT OF THE NEUROLOGICALLY-IMPAIRED FULL TERM INFANT

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To prevent handicaps we must recognize them to consider what may have caused them, and how best to deal with them in the newly born infant.

Slide 1

THE IMPORTANCE OF A NEONATAL NEUROLOGICAL EXAMINATION

1. Does it measure function above subcortical structures?
2. Are abnormal findings reliable indicators of future neurologic abnormality?

The clinical neurologic examination of the neonate has evolved slowly and has its detractors who see in it a tool with too many limitations for significant clinical implications in regard to either the assessment of higher nervous system activity or prediction of long-term outcome.

I propose to spend the very limited time available to us in this session today to demonstrate how I use the clinical examination of the full term neonate. This hardly approaches the question of the examination of the premature infant, though many of the principles are the same.

Slide 2

RISK FACTORS

1. Antenatal
2. Perinatal
3. Genetic

The history remains a most important part of any evaluation. But besides factors relevant to the teratogenic influences to which the pregnant mother may be exposed, the events during labour and delivery constitute a most important set of causes of insult to the CNS either in the form of hypoxia, ischemia, or physical traumatic injuries to the brain, the spinal cord, the brachial plexus, or peripheral nerves.

Slide 3

PREDICTORS FOR FUTURE CEREBRAL PALSY(1)

NEONATAL ABNORMALITY	RISK WEIGHTING
Diminished cry for more than one day	21 X
Need for gavage feeds	16 - 22 X
Diminished activity for more than one day	19 X
Weak or absent suck	14 X
Abnormality in tone	12 - 15 X

When we assess the neonate after birth, the following factors have been considered to increase the risk for cerebral palsy, by Nelson and Ellenberg.(1) Most of this information will only be available to the examiner if he/she takes the time to ask the nursing staff about the infant's cry, feeding behaviour, sucking and level of activity.

Slide 4

SPECIFIC SYMPTOMS OF NEUROLOGICAL DYSFUNCTION

1. Apathy
2. Apnea
3. Coma
4. Facial Palsy
5. Floppiness
6. Paralysis - Limb(S)
7. Poor Feeding
8. Progressive Macrocephaly
9. Seizures

It is important to consider specific symptoms of neurologic dysfunction.

Changes in level of arousal, such as apathy and coma, may be symptoms of systemic dysfunction, metabolic or cardiovascular, affecting brain function. Similarly, nonconvulsive apnea, seizures and floppiness may also reflect effects of primary metabolic derangements or cardiovascular disorder.

Changes in level of consciousness seriously affect the ability of the examiner to reach conclusions about the infant's cranial nerve function, including visual skills. Such derangements in level of arousal are a hallmark of hypoxic ischemic encephalopathy.

Slide 5

RATIONALE OF NEUROLOGICAL EXAMINATION OF THE NEONATE

1. The gestational age
2. Neurologic versus somatic maturation
3. Craniosomatic-facial proportions
4. Patent fontanelles and thin calvarium
5. Sleep states
6. Making primitive reflexes work for you
7. The autonomic nervous system's function

In examining the newborn, it is only logical to correct for the gestational age in measuring the performance against our expectations. I will not dwell on the use of the somatic parameters of gestational ages as compared to measures of tone initiated by the French neurologists and popularized by Dubowitz.(2) Suffice it to say that when somatic maturity far outstrips the infant's tone (on neurologic score) on the Dubowitz scale, CNS disorder should be seriously considered.

The patent fontanelles allow a clinical measure of increased intracranial pressure. This has been utilized presently in ICUs to have fontanometers continuously measure change in ICP.(3) The thinness of the calvarium certainly allows transillumination to detect lateralized cerebral atrophy or more diffuse structural changes such as hydranencephaly or hydrocephalus.

I heavily rely on item 6: Making the primitive reflexes work for you. This occurs at two levels.

First, at an interpretive level you analyse the primitive reflex in terms of its neuroanatomic and physiologic substrates and deduce the normality of its subset of parts: The 5th cranial nerve carrying the sensation from the face, the 7th cranial nerve allowing the infant to move its mouth towards the tactile stimulus, and the 11th cranial nerve allowing turning of the head to that side, in a rooting reflex.

Slide 6

STATE	EYES	RESPIRATION	CRY	MOVEMENTS	REFLEXES
I	C	R (Periodic)	D	O	+++
II	C REM Bursts	IR	O	+	O
III	O½	V	O	O±	+
IV	O	V	O	+	++
V	O	V	+	+	++

You often use the Moro reflex to deduce normal innervation of all 4 limbs and proprioceptive sensory input from the neck to elicit it.

Second, we use one complex function, specifically, sucking, to test other systems such as vision or hearing, or the ability of the infant to become habituated to repetitive stimuli in these modalities.(4)

Finally, the autonomic system is of great use in the neonate in assessing integrity of function. The early stages of anoxic encephalopathy may be characterized by sympathetic discharge with brachycardia and dilated pupils.(5) As the encephalopathy progresses, the pupils may become constricted, not dilating to darkness and reflecting parasympathetic tone. Respiration and temperature regulation are also parameters of autonomic nervous system integrity.

I am quite interested in the concept of sleep states, and the sleep/wake cycling of the infant as a predictor of its normality at levels including the subcortical and cortical structures.

It is possible that in the future development of on line computerized bedside monitoring to evaluate CNS function, especially in the full term and premature infant restricted by life support systems from participating in the traditional clinical assessment, will become a reality.

Slide 7

ANCILLARY TESTS

1. EEG - Routine
   - Prolonged - Polygraph
2. Cerebral evoked responses (Signal Averaging)
   - AVR
   - VER
   - SSR
3. CTT - Transaxial, coronal, saggital
4. Ultrasound
5. Motor Nerve Conduction Velocity
6. EMG
7. Lumbar Puncture (opening pressure)
   Cells and Protein
8. Skull x-ray

Certainly the neuro-investigative procedures available to us at present represent a new dimension of assessment, but are beyond the scope of today's discussion.

(A 20-minute videotape was presented which compared the examination of the full-term infant with that of an infant who looked quite normal at birth but had hydranencephaly.)

In conclusion, then, we document the integrity of the CNS function on physical examination in these 10 areas:

Slide 8

DOCUMENTATION OF THE NEUROLOGICAL EXAMINATION

1. State of arousal
2. Habituation and attention - Modality specific
3. Tone
   - a) Passive (Axial)
   - b) Active (Peripheral)
     - Proximal
     - Distal
4. Motor Movements
   - a) Spontaneous (Symmetry)
   - b) Elicited (Amplitude)
     (Power)
5. Cranial Nerve Examination(s)
6. Autonomic Nervous System
7. Sensation
8. Primitive Reflexes
9. Deep Tendon Reflexes
10. Transillumination

But the examination may be normal, and the history indicates increased risks for future dysfunction of CNS. This infant will need close monitoring so that early intervention may be initiated if an abnormality is later confirmed.

Slide 9

PREDICTION OF FUTURE NEUROLOGIC ABNORMALITY

1. Gross Abnormality
   Selective definite future deficit
   Diffuse definite future deficit
2. Definite Neurologic Abnormality 16% Later CP
   (Combinations of deficits) 50 X

3. Correlation in Context of Underlying Neuropathology	
Ex.: Neonatal Seizures	
a) Hypocalcemia	100% normal
b) Malform, asphyxia	0% normal
4. "Abnormal Tone"	Transient Dystonia
5. Normal examination?	Other Studies?

There may be subtle neurologic dysfunction on examination. Long-term outcome in these cases is not clear but may relate to clumsiness, school problems, or mild mental deficits.

No one will have difficulty with the gross type of abnormality when present on examination.

Slide 10

PATTERNS OF ABNORMALITY

1. Risk History, Normal Examination
2. Subtle Neurologic Dysfunctions
    a) Discrepant somatic-neurologic maturity scores
    b) Segmental tone abnormality
    c) Poor attention habituation in all modalities
3. Gross Abnormality
    a) Apathetic
    b) (Hemiparetic) - paretic
    c) Floppy
    d) Macrocephalic/microcephalic
    e) Abnormal transillumination
        - unilateral
        - see-through
    f) Abnormal cranial nerve

In predicting future outcome, the pattern of abnormality, selective or diffuse; the presence of more than one abnormality, increasing risks for poor outcome;(1) and the underlying neuropathology producing the manifestations in the neonatal period, should be carefully considered. Seizures 2° to malformations have a 0% of normal outcome. Those 2° to asphyxia have perhaps a 10-20% normal outcome as opposed to almost 100% normal outcome if hypocalcemic seizures are treated promptly.

I was asked to make recommendations relevant to methods to decrease or prevent handicap. I see these at five different levels.

First, at a genetic level. How many infants with a urea cycle defect have died undiagnosed! The parents have further children who survive but with mental retardation even with treatment.

Second. Identification of teratogenic influences on CNS development in utero. I have many infants with corpus callosal agenesis, megalencephaly, etc., with mild to severe mental delays and no one seems as yet to know the cause - only that it is likely to be environmental.

Third. The period of labour and delivery remains the period in which the frequency of insults to the CNS is most frequent. We need two developments at least, in this area:

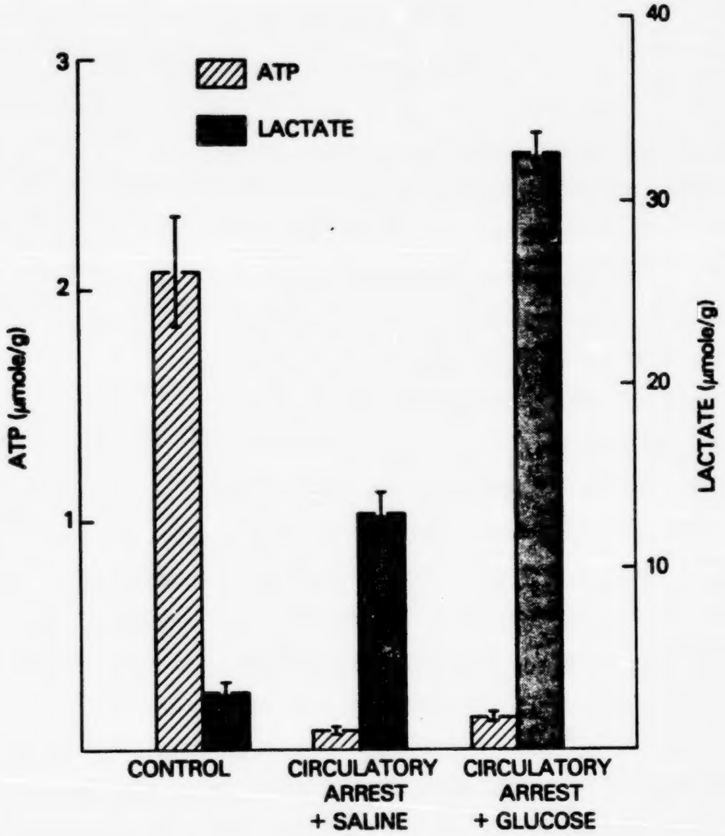
(a) Better and direct monitoring of CNS function during labour. This may be at neurophysiologic EEG level or cerebral blood flow perhaps, to correlate with changes in fetal heart rate or pH sampling.

(b) Research is sorely needed in the areas of treating acute CNS insult. Perhaps in no other areas of medicine can the physician say he is there when the insult is being sustained to the brain.

Animal studies have clearly shown that the early administration of steroids before the insult protects the brain whereas no protection occurs after the insult has occurred. But long-term effects on the CNS of early steroid medication have caused concern.(7)

Slide 11

Myers Appeared in Lactic Acid Accumulation as Cause of Brain edema, Advances in Perinatal Neurology, p. 85, 1979



Brain tissue concentrations of adenosine triphoshate (ATP) and lactate in food-deprived control animals and in animals exposed to 10 minutes of circulatory arrest following pretreatment with saline or glucose infusions (Myers and Yamaguchi 1976).

Other studies have shown that the animal pretreated with glucose infusion accumulates greater amounts of lactate and is left with lower amounts of energy-rich compounds in the CNS tissue than the animal pretreated with saline or the control who was starved prior to the insult.(8) This is startling and makes it obvious that much needs to be done in this area of brain resuscitation.

Slide 12

PRINCIPLES OF MANAGEMENT

1. Anticipation and Prevention
2. Early Resuscitation
3. Appropriate Diagnosis
4. Prevention of Recurrent Episodes
5. Management of Seizures
6. Treatment of Cerebellar Edema
7. Treatment of Intraventricular Haemorrhage?

Fourth. The infant has sustained an insult; in itself this results in a depressed CNS. It has been shown that the subsequent insult may lead to the irreversible injury.(9,10) The insulted infant who develops neonatal seizures or apneic episodes often experiences a lag of time between onset of these secondary and debilitating complications and onset of treatment for seizures and apnea. I have seen brief 10-20 second pauses in respiration cause drops of transcutaneous PO_2 to below 20 Torr. We stimulate the infants in the traditional manner, and they appear to initiate breathing again, but the $TcPO_2$, you may find as we have, does not rise above 30 Torr for quite a few minutes sometimes. In fact, these infants appear to spiral down into a cluster of apneic episodes after the first, leading finally to arrest and need for dramatic resuscitation.(11)

Greater awareness of these effects and a change in our response time and present strategy is necessary. Should we wait before we treat - or should be develop risk scores for seizures, apnea, etc., and then use anticipatory treatment before the 2° event occurs?

Fifth. Methods of long-term and early rehabilitation of the insulted CNS at high risk remain in need of research and clarification.

Slide 13

BONDING FACTORS

1. "Early" parent-infant physical contact
2. Parental minimal motivation and mental health factors
    a) Planned parenthood
    b) Family unit integrity
    c) Socio-economic factors
3. Integrity of a critical minimum of infant responses
    a) Visual fixation and eye contact
    b) "Cuddling"
    c) Quickness to console

Even bonding is affected by the presence of a handicap in an infant who is most in need of appropriate parenting!

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APPENDICES

A. Recommendations of Speakers and Moderators

B. Recommendations of Participants

C. Program Planning Committee

D. Steering Committee

E. Moderators

F. Speakers

G. Biographies

H. Participants

I. Program of the Third Symposium

APPENDIX A

RECOMMENDATIONS OF THE SPEAKERS AND MODERATORS

SYMPOSIUM ON HANDICAPPING CONDITIONS OF PRENATAL AND PERINATAL ORIGIN

OCTOBER 1981

Introduction

An important objective of the symposium was to generate recommendations which could be used as a focus for future activities in the prevention of handicapping conditions in the province of Alberta. Although the primary focus of the symposium was the prevention of cerebral palsy and brain damage, the recommendations deal with a much broader spectrum of prevention. The appropriate government agencies will be made aware of the recommendations.

The following are the recommendations generated by the speakers and moderators of the symposium.

Handicap Register

1. Establish a register of children with handicapping disorders, and devise mechanisms to ensure early reporting, and continuing contact with such children and their families, to ensure that they receive the help they require from resources which are available in the community.

Research into Prevention of Major Handicaps

2. Actively support research into strategies to prevent major handicapping disorders which are identified in the province.

Provincial Advisory Committee on Prenatal and Perinatal Care

3. Establish an advisory committee to the ministries concerned with health to recommend:

    a. Measures to reduce the high incidence of birth asphyxia in Alberta.

b. Minimal standards of obstetric and perinatal care for all Alberta hospitals.

c. Standards for family-centred maternity care in Alberta hospitals.

d. Necessary changes in university and postgraduate programs for health care professionals to ensure optimal reproductive care.

Immunization Standards

4. Standardize requirements for immunization of children in Alberta.

Developmental Screening Programs for Mild Developmental Delay

5. Establish a detection program to ensure early intervention for children with "mild" mental retardation.

Pregnancy Allowance

6. Recommend to the Federal Government that the family/child allowance be paid to mothers on confirmation of pregnancy and until the child's 17th birthday, so as to provide a financial resource for mothers in their pregnancies.

Dr. Graham Chance
October 30, 1981

APPENDIX B

RECOMMENDATIONS OF THE PARTICIPANTS

SYMPOSIUM ON HANDICAPPING CONDITIONS OF PRENATAL AND PERINATAL ORIGIN

OCTOBER 1981

Introduction

Each participant at the symposium was invited to submit recommendations on the prevention of handicaps which were appropriate either for the province of Alberta or the national scene. It was requested that the recommendations be relevant to the presentations and recommendations made by the speakers. The recommendations were reviewed by the Program Planning Committee and the following list compiled.

Recommendations

Prenatal Smoking and Alcohol

1. Develop a program for preconceptual and prenatal alcohol and smoking reduction and control. There is a need for planned, co-ordinated programs. A model is available in Nova Scotia and Saskatchewan.

Education

2. Educate key health professionals in newborn resuscitation through the Provincial Perinatal Education Program. At present there is a need for a comprehensive educational program and an at risk criteria review.

Follow-up

3. Establish linkages between ICN personnel and perinatal follow-up programs. There needs to be standardized screening. Community

health nurses should include DDST, height, weight and head circumference in screening.

Integration

4. Integration of follow-up activities into perinatal programs.

Early Detection

5. Assessment of newborn and infant screening for early detection of handicapping conditions.

Native Groups

6. Epidemiological evidence is needed to support suspicion of high morbidity and mortality rates in native groups. Therefore, there is a need for a long-term study of several native and Metis communities.

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Appendix E

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Appendix G

BIOGRAPHIES

The Honourable Bob Bogle is the Minister of Alberta Social Services and Community Health and is MLA for the Taber-Warner Constituency. Mr. Bogle received his secondary education at the University of Lethbridge and Sir George Williams University, and, prior to his election, served as teacher, high school administrator and businessman in Southern Alberta.

Dr. Graham Chance is Director of Nurseries at St. Joseph's Hospital and Professor in the Department of Paediatrics, Obstetrics and Gynaecology, University of Western Ontario, London, Ontario. A paediatric neonatologist, Dr. Chance has been particularly interested in infant nutrition, endocrinology and metabolism. He has been involved extensively in research, and his work in this field has been widely published. Dr. Chance is actively involved in many professional societies and academic committees. He has been responsible for instituting a regional perinatal program in Ontario.

Dr. Husam Z. Darwish is Associate Professor of Paediatrics and Clinical Neurosciences at the University of Calgary Medical School. Dr. Darwish's interests are in neonatal neurology, behavioural neurology and electrophysiology. His current research includes the study of the sleep states in premature infants with apnea, and the efficacy of pemoline in hyperactive children. Dr. Darwish is paediatric neurologist on staff at the Alberta Children's Hospital and Foothills Hospital.

Dr. Neil N. Finer is Director, Neonatal and Paediatric Intensive Care, Royal Alexandra Hospital, and Associate Professor of Paediatrics, University of Alberta, Edmonton. Dr. Finer's activities focus on the care of the critically-ill neonate and paediatric critical care medicine. He is currently involved in the long-term follow-up of the asphyxiated fullterm neonate, and is also researching the effects of vitamin E on ventilated premature infants in preventing retrolental fibroplasia.

Dr. A. Mervyn Fox is the Associate Professor of Paediatrics, University of Western Ontario, and Medical Director, Thames Valley Children's Centre, London, Ontario. Dr. Fox's activities are focussed on developmental and behavioural paediatrics. He has a particular interest in parent management and child/family assessements of developmental disorders. Follow-up and anticipatory guidance with high risk infants and outcome studies in cerebral palsy are current research interests. Dr. Fox is a member of the Board and Grants Committee, Conn Smythe Research Foundation for Crippled Children and the Professional Advisory Committee, Easter Seal Society, Ontario.

Dr. William K. Frankenburg is Director of the John F. Kennedy Center, and Professor of Paediatrics and Preventive Medicine at the University of Colorado, Denver, Colorado. Child development is Dr. Frankenburg's specialty, and he is world renowned as the orginator of the Denver Developmental Screening Test. His current research is directed towards the early identification of children with significant developmental problems and an evaluation of the efficacy of early intervention.

Mrs. Agnes Hall is the Chairman, Manitoba Community Task Force on Maternal and Child Health. She is a community volunteer and fund raiser who has held positions on the board of the Canadian UNICEF Committee and as Chairman of YWCA Building Fund in Winnipeg. Mrs. Hall is particularly interested in women's health issues, and is currently teaching a "Special Needs" class for pregnant teenagers, sponsored by the Winnipeg School Board.

Dr. Robert Haslam is Professor and Head of the Department of Paediatrics at the University of Calgary. Dr. Haslam's specialty is in the field of paediatric neurology. His current research relates to the investigation of pharmacological methods of treatment for childhood epilepsy and learning disabilities, as well as examining the use of C.T. scanning in children with learning disabilities. Dr. Haslam was the Chairman of the two previous Symposia and has maintained a major interest in preventive activities.

Dr. Warren Hindle is Director of Ophthalmology, Alberta Children's Hospital and a Paediatric Ophthalmology Consultant at the Alberta Children's Hospital and Foothills Hospital, Calgary, and also an Associate Professor, University of Calgary, Faculty of Medicine. Dr. Hindle has a particular interest in ophthalmic diseases of children and in retinopathy of prematurity. He is currently involved in research of retrolental fibroplasia, the course and sequelae of this disorder.

Mrs. April Katz is Chief of Compliance, British Columbia Human Rights Branch, Victoria. She is also an Executive Member, Community Task Force on Maternal and Child Health, Manitoba. Mrs. Katz has served as Counsel for many organizations, including Native Communities, the Coalition of Provincial Organizations of the Handicapped, Women's Centre and Law Reform Commission. Professionally, Mrs. Katz has been active on the Executive of the Family Law Subsection of the Manitoba/Canadian Bar Association, the Manitoba Association of Rights and Liberties, Association of Women and the Law, and the Legal Aid Lawyers Association.

Ms. Jo Anne M. Mahood is a Speech Pathologist at Glenrose Hospital, Edmonton. Ms. Mahood's major interest centres on non-speech communication development in the young severely physically handicapped child, as well as parent training for this population. Her clinical activities include participation in the Neonatal Follow-up Clinic (assessment and treatment) and diagnosis, and treatment of children under three years "at risk" of not developing functional speech.

Dr. Peter W. Nathanielsz is Director, Laboratory of Fetal Physiology, Department of Obstetrics and Gynaecology at Harbour/UCLA Medical Centre, Torrance, California. Dr. Nathanielsz is a specialist in fetal endocrinology and growth, and he is currently involved in an experimental investigation of fetal growth in the pregnant sheep and monkey models. He is the author of "Fetal Endocrinology - An Experimental Approach".

Mrs. Shirley Post is the Executive Director of the Canadian Institute of Child Health, Ottawa, and a founding member. Mrs. Post has a nursing education and administration background, and her field of interest is in the realm of maternal and child health, with current emphasis on childhood accidents and safety. She has lectured and published extensively in Canada on a range of topics, including family-centred maternity and newborn care.

She is currently the President of the International Association for the Care of Children's Health.

Dr. Sally Provence is a Professor of Paediatrics, Yale Child Study Centre and the former Director, Child Development Unit, Yale Child Study Centre, New Haven, Connecticut. Dr. Provence specializes in the field of child development and directs her clinical activities to diagnosis and treatment of developmental health problems of infants and young children. She has a particular interest in parent-child relationships and is currently researching the effectiveness of early intervention into problems of child mental health.

Dr. Bob Richards has been director of Obstetrical Perinatal Services at the Royal Alexandra Hospital in Edmonton since 1976. He graduated from the University of Western Ontario, London, Ontario in 1962. He received his Fellowship in 1968 and was the McLaughlin Travelling Fellow, Oxford, 1969-1970.

Dr. Charlene N. T. Robertson is Medical Director and Paediatrician, Department of Paediatrics, Glenrose Hospital and Associate Professor of Paediatrics, University of Alberta, Edmonton. Dr. Robertson's range of interests include child neurology and developmental medicine. She is currently researching the long-term follow-up of neonatal intensive care graduates and the interpretation of changing neurological signs in young children. Dr. Robertson is also the Director of the Neonatal Follow-up Program, Northern and Central Alberta Perinatal Program.

Dr. David Rush is Associate Professor, Public Health (Epidemiology) and Paediatrics, Faculty of Medicine, Columbia University, New York, N.Y. At this time Dr. Rush's activities are focussed on the effects of maternal health, nutrition and social class on the developing fetus. The long-term effect of maternal smoking on the fetus is a current research interest. Dr. Rush is President of the Society for Epidemiological Research, 1980/81, and the Associate Attending Paediatrician, Babies and Harlem Hospitals.

Dr. Harvey B. Sarnat is Associate Professor of Paediatrics (Neurology) and Pathology, University of Calgary Faculty of Medicine, and Paediatric Neurologist and Neuropathologist at the Alberta Children's Hospital, Calgary. Dr. Sarnat's interests are in neuromuscular disease, particularly congenital myopathies, and neonatal neurology. His current research is the ontogenesis of striated muscle and developmental disorders of muscle, in terms of comparative neuroanatomy and evolutionary theory.

Dr. Reginald S. Sauve is Associate Professor, Department of Paediatrics and Community Health Sciences, University of Calgary, and Director, Perinatal Follow-up Program, Alberta Children's Hospital, Calgary. Dr. Sauve's activities are focussed on clinical neonatology and perinatal follow-up. His current research involves monitoring of the process and outcome of perinatal care.

Professor Sir Peter M. Tizard is Professor of Paediatrics, University of Oxford, John Radcliffe Hospital, Headington, Oxford, England. Professor Tizard's major interests concern the newborn and paediatric neurology. He is currently engaged in research into the origins of cerebral palsy, as well as cerebral blood flow in the newborn. His clinical activities comprise general and neonatal paediatrics. Professor Tizard is the President-Elect of the British Paediatric Association.

Dr. Joe Watt is the Associate Director of the Rehabilitation Department, Glenrose Hospital, Edmonton. Paediatric physical medicine and rehabilitation are the fields in which Dr. Watt centres his activities. He is currently researching the effects of inhibitive casting on the cerebral palsy child.

Dr. Jonathan S. Wigglesworth holds the position of Reader in Paediatric Pathology, Institute of Child Health, Hammersmith Hospital, London, England. Dr. Wigglesworth is particularly interested in the interaction of pathological processes with normal growth and development, and his activities are directed to the organization of a perinatal pathology service as a practical aid to parents and clinicians. The control of fetal lung development and the pathophysiology of lung hypoplasia, and the pathophysiology of perinatal brain damage, are two current areas of research. He is a member of the Academic Board of the British Paediatric Association and the Scientific Advisory and Pathology Committee of the Royal College of Obstetricians.

Appendix H

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Richardson, Blair
Richle, Gail
Richmond, Joy
Roberts, Adele
Robertson, Gail
Robinson, Betty
Robinson, Dr. Geoffrey

Ross, Janet
Rowledge, Davis
Roy, Sharon

S. Sabey, Marie
Salkin, Brenda
Salt, Frances
Saunders, Theresa
Schafer, Valerie
Schmidt, Barbara
Schmidt, Dr. Brenda
Schmidt, Linda
Schmitt, Joan
Schooler, Shirley
Scobie, Robin
Siben, Con
Silkie, Kathleen
Sirois, Pat
Smith, Dr. Margaret
Solem, Paddi
Soubiran, Dawn
Sparrow, Heather
Spavor, Louis
Spot, Isabel
Stangier, Sheila
Stensland, Dave
Stoddart, Claudette
Stow, Chris
Stuart, Elisabeth
Sutherland, Heather

T. Taylor, Elizabeth
Tenove, Patricia
Teran, Dr. J.J.
Thuillier, Erma

Toane, Dr. E.B.
Tomkins, Dr. Lynn
Toner, Mary Louise

U. Unraw, Henry

V. Van Laare, Evelyn
Vijayakumtar, Dr. C.P.

W. Waddell, Kathy
Wallace, Hollie
Wallsten, Dacia
Walman, Barbara
Ward, J.F.
Ward, Dr. Tom
Warwick, Marian
Wedel, Dr. Robert
Whyte, Dr. Lillian
Willis, Connie
Winski, Linda
Wolter, Janice
Wong, Dr. Lei Ching
Woodhams, Wendy
Wooley, Sue
Woronuk, Sharon

Y. Young, Marilyn

Z. Zackary, Marlene
Zerb, Sandra

Appendix I

PROGRAM FOR THE THIRD SYMPOSIUM ON THE PREVENTION OF HANDICAPPING CONDITIONS OF PRENATAL AND PERINATAL ORIGIN

OCTOBER 28/29/30 1981

GLENROSE HOSPITAL, EDMONTON, ALBERTA, CANADA

Sponsors: Alberta Social Services and Community Health
Canadian Institute of Child Health
Glenrose Provincial Hospital

WEDNESDAY 28th, OCTOBER

	Topic	Speaker	Moderator
1930 - 2030 Public Address	Welcome	Lloyd Wilson Chairman, Glenrose Hospital Board	
	John T. Law Lecture - Newborn Care and the Prevention of Cerebral Palsy	Dr. Peter Tizard Oxford, England	Dr. William Cochrane President, Canadian Institute of Child Health Toronto, Ontario
2030 - 2100	Questions and Answers		

THURSDAY 29th, OCTOBER

Opening Ceremonies

	Topic	Speaker	Moderator
0830 - 0835	Welcoming Address	Lloyd Wilson Chairperson, Glenrose Hospital Board	
0835 - 0845	Report on Recommendations from the First and Second	Dr. Robert Haslam Calgary	
0845 - 0900	Opening Address	Honourable Bob Bogle Minister of Social Services & Community Health, Edmonton	Dr. Tom Paton Edmonton
Morning Theme - Brain Development			
0900 - 1000 Plenary Session	Factors Influencing Brain Development	Dr. Jonathan Wigglesworth London, England	Dr. David Schiff Edmonton
1000 - 1030	Nutrition Break		
1030 - 1130 Plenary Session	The Relationship of Maternal Cigarette Smoking During Pregnancy to Physical and Psychological Development at Age 5	Dr. David Rush New York, New York	Dr. David Schiff Edmonton
1140 - 1230 Concurrent Sessions	A. Intraventricular Haemorrhage and Hypoxic Injury	Dr. Jonathan Wigglesworth London, England	Dr. Phil Etches Edmonton
	B. Identification of High Risk Factors and Treatment of the Asphyxiated Full Term Neonate	Dr. Neil Finer Edmonton	Dr. Bob Richards Edmonton
	C. Development of High Risk Infants in the First Year of Life	Dr. Reginald Sauve Calgary	Dr. Charlene Robertson Edmonton
1230 - 1400	Lunch		
Afternoon Theme - Effects of Brain Damage			
1400 - 1500 Plenary Session	The Effect of Physiological and Pathological Changes in Oxygen Delivery to the Fetal Brain on Normal and Abnormal Brain Development	Dr. Peter Nathanielsz Los Angeles, California	Dr. John Parboosingh Calgary
1500 - 1530	Nutrition Break		
1530 - 1630 Plenary Session	Assessment of the Fetus at Risk	Dr. Bob Richards Edmonton	Dr. John Parboosingh Calgary
1640 - 1730 Concurrent Sessions	A. Early Sensori-Motor Intervention - Who, Why and How to Assess	Dr. Mervyn Fox London, Ontario	Dr. Gerard Kysela Edmonton
	B. Assessment of the Neurologically Impaired Full Term Infant	Dr. Husam Darwish Calgary	Dr. Marc Beaudry Edmonton
	C. Neurologic Development in the Human Fetus and Assessment of the High-Risk Neonate	Dr. Harvey Sarnat Calgary	Dr. Ehor Gauk Edmonton
	D. Ophthalmic Problems of Prematurity	Dr. Warren Hindle Calgary	Dr. Garry Grant Edmonton

FRIDAY 30TH, OCTOBER

Morning Theme - Early Recognition and Treatment

	Topic	Speaker	Moderator
0900 - 1000 Plenary Session	To What Extent Can We Prevent Mental Retardation	Professor Sir Peter Tizard Oxford, England	Dr. Tom Paton Edmonton
1000 - 1030	Nutrition Break		
1030 - 1130 Plenary Session	The Denver Approach to the Developmental Screening and Diagnosis of Infants and Pre-school Children	Dr. William Frankenburg Denver, Colorado	Dr. Tom Paton Edmonton
1140 - 1230 Concurrent Sessions	A. Advances in Assessment and Treatment of the Child with Cerebral Palsy	Dr. Charlene Robertson Dr. Joe Watt Edmonton, Alberta	Dr. Peter Tizard London, England
	B. Prediction of Communication Disorders	Jo Anne Mahood Edmonton	Dr. Robert Shea Edmonton
	C. The "High Risk" Concept Revisited	Dr. William Frankenburg Denver, Colorado	Doreen Paddon Edmonton
	D. Primary Prevention of Mental Illness in Infants	Dr. Sally Provence New Haven, Conn.	Dr. Allan Carroll Edmonton
	E. A Look at Family Centred Maternity Care - The Canadian Picture	Shirley Post Ottawa, Ontario	Dr. John Parboosingh Calgary
	F. Childbearing Families and The Law - The Manitoba View	Agnes Hall April Katz Winnipeg, Manitoba	Cathy Miller Edmonton
1230 - 1400	Lunch		

Afternoon Theme - The Family

1400 - 1530 Plenary Session	Family Relationships and Intervention	Dr. Sally Provence New Haven, Conn.	Dr. Allan Carroll Edmonton
1500-1530	Panel Discussion on the Family and Intervention		Dr. Allan Carroll Edmonton
1530 - 1600	Symposium Recommendations Concluding Remarks	Dr. Graham Chance London, Ontario	
1600 - 1630	Afternoon Tea and Farewells		

Please Note: The Symposium has been accredited for 10 hours of study time by the College of Family Physicians of Canada. Contact the Registration desk for accreditation note.

HS23J